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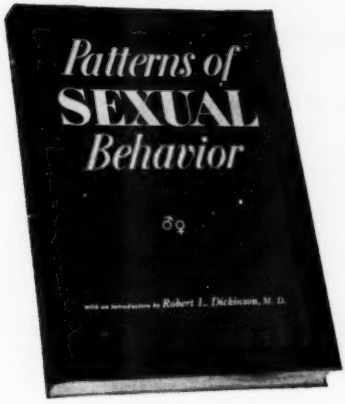

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INTELLECTUAL AND EMOTIONAL MAKEUP OF THE EPILEPTIC

FREDERIC T. ZIMMERMAN, M.D.
BESSIE B. BURGEMEISTER, Ph.D.
AND
TRACY J. PUTNAM, M.D.
NEW YORK

THE FIELDS of epilepsy and mental retardation have engaged our interest for a number of years because we feel that a study of these clinical conditions may throw light on the mechanism of conscious awareness in man. Both conditions appear related to us in the sense that epilepsy may be regarded basically as an acute interruption or disturbance of conscious awareness, while mental retardation manifests itself primarily as a chronic diminution of the state of conscious awareness.

Since man's mental functioning depends on the efficiency of his state of conscious awareness, it follows that a study of the intelligence of the epileptic shows some relation between the type or degree of disturbance in conscious awareness, i. e., the type of seizure, and the adequacy of mental function.

It is the purpose of the present paper to summarize five years of research on the relation of epilepsy, intelligence and personality, as indicated by various objective psychological tests, supplemented by extensive clinical data. It represents an effort to attack the problem of epilepsy as a whole—psychologically, as well as physiologically. These two approaches complement each other naturally and support our thesis that epilepsy is a disturbance of the total organism. As such epilepsy can be understood and treated only as an indivisible whole. Our data likewise throw further light on the controversial question of the "epileptic personality."

MATERIAL AND METHODS

Source of Material.—The material on which the study of the relation of epilepsy, intelligence and personality is based consists of 300 case records from the Neurological Institute, Columbia-Presbyterian Medical Center. The cases represent equal proportions of private and ward patients admitted to the hospital for observation and study and we believe the group constitutes a fairly good random sample of the general population. As a further check, a small series of patients from Vanderbilt Clinic was added, and their records show great similarity to the hospitalized group. Two hundred of our patients were adults. One hundred were children and adolescents.

Clinical Data.—While data covering many aspects of epilepsy were available in the case histories, only those factors which were considered to have a direct bearing on the psychosomatic

This research was made possible by grants from the Commonwealth Fund.

From the Department of Neurology, Columbia University College of Physicians and Surgeons, and the Neurological Institute of New York, Division of Child Neurology.

nature of the problem were extracted. The following information was recorded: (1) age of onset of seizures, (2) duration of epilepsy, (3) duration of attacks, (4) type of seizure, (5) frequency of seizures, (6) previous treatment, (7) duration of treatment, (8) personality traits and (9) history of familial epilepsy.

Psychological Tests.—The Stanford-Binet Intelligence Test, Form L, 1937 revision,[1] was administered to the 100 children and adolescents in our group. Ninety-eight of them received the Arthur Point Scale[2] or Merrill-Palmer[3] performance tests, and 58, the Rorschach ink blot test.[4]

The Wechsler-Bellevue Intelligence Scale[5] was given to the 200 adults. Rorschach records of 167 of our adult group were available.

Classification of Epilepsy.—The majority of previous investigators who have attempted to study the effect of epilepsy on intelligence have contented themselves with a loose grouping of cases under such headings as "organic" and "nonorganic." This procedure was based, no doubt, on the reasoning that a lowering of intelligence, if present at all, should be more clearly discernible in "organic" cases, and statistics reported in the literature bear out this assumption.

It is our feeling, however, that the use of such a dichotomy merely scratches the surface of the problem and leaves many differences in intellectual capacity and personality development obscured. We believe that these differences may be detected by a method of classification which utilizes finer degrees of differentiation, and that varying degrees of organic and emotional interference with mental functioning may more clearly be revealed if the type of epileptic seizure is considered.

We have therefore divided our patients according to the following types of seizure and etiology: (1) idiopathic petit mal, (2) idiopathic petit mal and grand mal, (3) idiopathic grand mal, (4) symptomatic epilepsy and (5) traumatic epilepsy. It was impossible to establish a clearcut diagnosis of psychomotor epilepsy in a sufficient number of cases to draw valid conclusions for a study of this sort. Furthermore, Lennox has already established in his study[5a] that patients with psychomotor epilepsy have a rather low intelligence quotient as a group.

Our findings are interpreted on the basis of the above classification, in keeping with our original hypothesis regarding the value of the type of seizure as a basis of intellectual and personality differentiation of epileptic patients.

RESULTS

Clinical Findings.—The 100 children and adolescents in our study consisted of 54 (54 per cent) males and 46 (46 per cent) females, ranging in age from 3 to 16 years. The average age for the group was 9 years 6 months, and the average duration epilepsy, 3 years 6 months, placing the average time of onset at 6 years. In our group of 200 adults, there are 110 (55 per cent) males and 90 (45 per cent) females, the ages ranging from 16 to 71 years, the average age for the group being

1. Terman, L. M., and Merrill, M. A.: Measuring Intelligence: A Guide to the Administration of the New Revised Stanford-Binet Tests of Intelligence, Boston, Houghton Mifflin Company, 1937.

2. Arthur, M. G.: A Point Scale of Performance Tests, New York, Commonwealth Fund, 1930.

3. Stutsman, R.: Mental Measurement of Preschool Children, with a Guide for the Administration of the Merrill-Palmer Scale of Mental Tests, Yonkers-on-Hudson, N. Y., World Book Company, 1931.

4. Rorschach, H.: Psychodiagnostik, Bern, Hans Huber, 1942.

5. Wechsler, D.: The Measurement of Adult Intelligence, Baltimore, Williams & Wilkins Company, 1939.

5a. Collins, A. L., and Lennox, W. G.: The Intelligence of 300 Private Patient Epileptics, A. Res. Nerv. & Ment. Dis., Proc. **26**:586-603, 1947.

28 years 4 months at time of testing. It appears of interest that the reported average time of onset of seizures was at the age of 19 years 10 months, and the duration of epilepsy eight years, five months, for the adult group. This age of onset seems rather late, especially in view of our findings for children, for whom the average time of onset was placed at 6 years.

The average duration of the grand mal attacks for 40 children was 11 minutes. No average time of duration of attack was computed for other types of seizures, because such factors as shortness of duration and uncertainty of time of onset do not lend themselves to the computation of reasonably valid averages.

According to our classification, the following frequencies of incidence are revealed:

	Children (100)	Adults (200)
Idiopathic petit mal	14	10
Idiopathic petit mal and grand mal	15	49
Idiopathic grand mal	42	86
Symptomatic epilepsy	8	29
Traumatic epilepsy	21	26

The effect of previous anticonvulsant treatment could not be evaluated accurately because the hospital records were inadequate in the recording of such vital data as type and amount of drug administered and duration of treatment before admission.

The number and nature of personality traits recorded, as well as the history of familial epilepsy, did not prove significant in our study. Many reported "personality traits" failed to find verification in individual Rorschach records of our patients or in clinical observations.

Results of Psychological Tests.—Children and Adolescents (Stanford-Binet Intelligence Test): On the Stanford-Binet test, the average intelligence quotient for our 100 children was 92.60 (standard deviation [σ], 22.00; average σ, 2.2). This quotient, which is the ratio of mental age to chronological age, represents the equivalent of 8 years 9 months in mental age, or a mental age which is 9 months below the group chronological age. While falling within the average range (90 to 109), the quotient is considerably lower than is expected of children in general, i. e., approximately 100. In this respect our results agree substantially with those of other investigators (Dawson and Conn,[6] Harrower-Erickson,[7] Fetterman and Barnes,[8] Somerfeld-Ziskind and Ziskind[9]).

In terms of percentages, 10 per cent of our group had an intelligence quotient of 110 or above, which is high average or superior, and 24 per cent had an intelligence quotient below 80, or within the ranges of borderline and defective intelli-

6. Dawson, S., and Conn, J. C. M.: The Intelligence of Epileptic Children, Arch. Dis. Childhood **4**:142-151, 1929.

7. Harrower-Erickson, M. P.: Personality Studies in Cases of Focal Epilepsy, abstracted, Bull. Canad. Psychol. A. **5**:19-21, 1941.

8. Fetterman, J., and Barnes, M. R.: Serial Studies of the Intelligence of Patients with Epilepsy, Arch. Neurol. & Psychiat. **32**:797-801 (Oct.) 1934.

9. Somerfeld-Ziskind, E., and Ziskind, E.: Effect of Phenobarbital on the Mentality of Epileptic Patients, Arch. Neurol. & Psychiat. **43**:70-79 (Jan.) 1940.

gence. Data of a report on epileptic children made by the New York City Board of Education in 1941 [10] yield the following information:

Elementary School Pupils	Attending School	Excluded from School (Home Instruction)
Intelligence Quotient		
Below 80	84 (19%)	49 (37%)
Above 110	52 (32%)	5 (4%)
Not stated	113	80
High School Pupils		
Intelligence Quotient		
Below 80	7 (23%)	3 (25%)
Above 110	9 (30%)	3 (35%)
Not stated	14	6

An explanatory note added to the report of the Board of Education states: "By review of questionnaire from two boroughs on children attending school, judging probable mental level by information supplementing whatever was stated with respect to I. Q., in a total of 240 pupils I. Q. of less than 80 was found in 60, or 25 per cent."

Intelligence quotients in our study of epileptic children agree well with those of children with seizures recently attending the New York city public schools at the

TABLE 1.—*Number, Age and Intelligence Quotient of Children and of Adolescents in Our Classification of Epilepsy*

Classification	No.	Average Age, Yr.	Average I. Q.
Entire group	100	9½	92.60
Idiopathic petit mal	14	8	105.50
Idiopathic petit and grand mal	15	11	91.50
Idiopathic grand mal	42	8½	91.25
Symptomatic epilepsy	8	10	89.00
Traumatic epilepsy	21	10	89.00

lower intelligence quotient levels, but our total distribution is positively skewed, when compared with the data of the Board of Education or those for the population at large.

In most investigations reported in the literature which are concerned with the intelligence quotients of epileptic children, the quotient has been derived by combining the individual scores of patients representing all types of convulsive disorders. If, on the other hand, data are considered according to classification based on type of epilepsy, as in our present study, interesting differences in intelligence quotients appear among the various subgroups which are concealed in the combined quotient. Table 1 gives the number of children and adolescents included in each of our subgroups and their average intelligence quotient.

It may be noted from table 1 that the intelligence quotient for the children with petit mal epilepsy is 105.50, a score which is definitely above the average of the population at large. Quotients for the other categories range from 91.50, for idiopathic grand and petit mal, to 89.00, for traumatic epilepsy and symptomatic epilepsy, and are below the group mean of 92.60. Although the numbers of children in these subgroups are relatively small, there is a striking difference (13.90 points) between the intelligence quotient of the petit mal group and the intelligence quo-

10. Epileptic Children, Report of New York City Board of Education, New York, 1941.

tients of children in all four of the other categories. According to Terman and Merrill,[1] the maximum difference expected by chance in a normal population is 12.16 points, or four times the probable error of the group mean. When their criterion is used, our findings point in the direction of a significant difference, provided our sample is a typical one, with the petit mal group far superior to patients with the severer type of epileptic seizure.

Table 2 shows the differences in performance test scores when division into subgroups is made according to our classification of epilepsy.

As may be seen from table 2, children and adolescents with idiopathic petit mal seizures show a performance test average (110) which is well above the average for the epileptic group (89) and which is also higher than the mean score for children of this age, i. e., approximately 100. In terms of ability, the score for the petit

TABLE 2.—*Number and Performance Test Scores of Children and Adolescents in Our Classification of Epilepsy*

Classification	No.	Average Performance Quotient
Entire group	98	89
Idiopathic petit mal	14	110
Idiopathic grand and petit mal	15	88
Idiopathic grand mal	40	84
Symptomatic epilepsy	8	78
Traumatic epilepsy	21	73

TABLE 3.—*Number, Age and Intelligence Quotients of Adult Groups According to Our Classification of Epilepsy*

Classification	No.	Average Age, Yr.	Average I. Q.
Entire group	200	28	100.35
Idiopathic petit mal	10	29	108.70
Idiopathic petit and grand mal	49	29	103.54
Idiopathic grand mal	86	26	98.50
Symptomatic epilepsy	26	35	98.13
Traumatic epilepsy	29	26½	92.52

mal group would indicate high average skill in handling this kind of performance material.

In contrast to this capacity, ratings for our other subgroups of epileptic children of approximately the same chronological age fall below these two means and range from 73 to 88. Results on both verbal and motor tests therefore indicate striking differences in ability to make high scores between children with seizures of idiopathic petit mal and those in all our other categories, whose scores in each instance fall below the mean of the entire group. Both verbal and motor test averages for the combined group are also below those expected of children of this age in general.

Wechsler-Bellevue Intelligence Test: Table 3 shows that the highest intelligence score is found in the adult group with idiopathic petit mal seizures, as it was among the children in our study (table 1). This rating of 108.70 is near the lower end of the high average range and well above the mean (100.35) for the combined group (σ, 17.20; average σ, 1.08). The score of 103.54 for the group having idiopathic petit and grand mal seizures is also above average, but ratings for the groups with

idiopathic grand mal, and symptomatic and traumatic epilepsy are below the combined group average, as they were among the children and adolescents, being 98.50, 98.13 and 92.52, respectively. The scores for the combined idiopathic subgroups are higher than those for the groups with symptomatic or traumatic epilepsy, as might be expected from the nature of the pathology in the latter categories. These results are in keeping with the findings of other investigators who use the "nonorganic" and "organic" classifications, and it is generally conceded that when an organic condition is severe, interference with mental functioning can more readily be discerned on intelligence tests than when it is less acute (Wechsler,[5] Arieff and Yacorzynski,[11] Lennox and Collins [12]).

Analysis of results also necessitated consideration of the possible relationship of the frequency to the severity, as well as the type, of epileptic seizure. An intensive statistical study of the largest group of 40 children having idiopathic grand mal epilepsy has been made, and results will be reported in another publication. Evidence indicates, however, that a lower correlation exists between frequency, severity or duration of epilepsy and the intelligence quotient than between the quotient and age of onset, the intelligence quotient being higher when the onset of seizures appears late than when it appears early in the child's life. We believe our data demonstrate that the nature of the disorder is of greater importance than any of the above factors in determining the intelligence quotient among our group of children and adolescents.

Rorschach Findings: Rorschach ink blot material was available on 167 of our 200 adult patients. We are not reporting our data on the children's Rorschach tests here because of differences in age level and the small numbers of subjects in some of our groups.

The Rorschach test is a projective technique which sheds light on the dynamics of motivation and the mechanisms underlying behavior. It consists of a series of 10 cards in which the subject is required to interpret unstructured ink blot material, and the approach is much the same as that used in judging an artist's painting. It is well known that in the judgment of an artistic production a large subjective factor enters in, and that a person's reaction is influenced much more by his own tastes and values than by the objective criteria of what the artist is trying to do and whether or not he has succeeded. This subjective factor operates even more potently in ink blot material, and the subject puts whatever meaning there is into the blots. Such a technique makes it possible to gain insight into the clarity of a subject's thought and of his perceptions, his creativity and his approach to problems generally.

The Rorschach test has been standardized on large numbers of people and is a valid instrument not only for revealing personality mechanisms, but for estimating the subject's intellectual level. It is also very sensitive to impairment of intellectual functioning on an emotional basis and to interference with thought processes having an organic origin, such as arises from head injury, encephalitis and removal of the frontal lobes. For this reason, we divided our Rorschach data for adult epileptic patients according to the etiological factor, as we did in the case of the intelligence test material.

11. Arieff, A. J., and Yacorzynski, G. K.: Deterioration of Patients with Organic Epilepsy, J. Nerv. & Ment. Dis. **96**:49-55, 1942.

12. Lennox, W. G., and Collins, A. L.: Intelligence of Normal and Epileptic Twins, Am. J. Psychiat. **101**:764-769, 1945.

Our group of patients with idiopathic petit mal epilepsy is small but shows more productiveness and relatively better social and emotional adjustment than any of our other groups (chart 1). The records reveal slightly less creativity than is desirable at this level of intelligence and a less favorable balance between capacity and production than might be expected of well adjusted adults. Some emotional instability is apparent, and a rather immature intellectual approach to problems is customarily employed. There is evidence that the inner life is less rich in our group than it is in samples of other adult populations, and that less inner stability exists. Interest in social contacts is evident, but an egocentric, impulsive approach characterizes the response, less than the desired amount of social adaptability and conformity being apparent. These Rorschach data are in striking contrast to those obtained from a group of patients in the early stages of multiple sclerosis, in which a much more adaptive type of social response was indicated.[13]

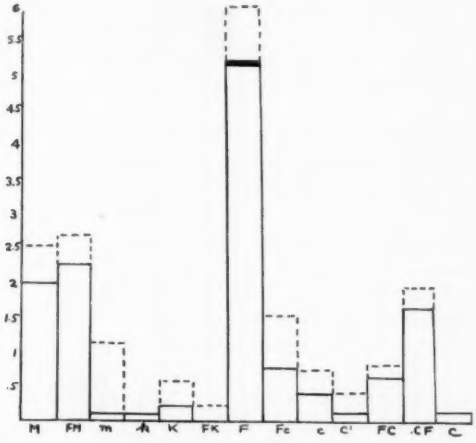

Chart 1.—Rorschach patterns for our group of adults with idiopathic petit mal.

In this chart, and in chart 2, the main responses are indicated by solid line rectangles; additional responses, by dotted line rectangles, and minus forms, by black areas. Our petit mal patients have the best Rorschach protocols of any of our epileptic groups, although limited productiveness is reflected by records with an average of 15 responses, a limited number of M's (human movement) and a discrepancy between capacity and drive (W : M ratio, 3.2 : 1, whereas the ideal ratio is 2 : 1 [Klopfer [14]]).

Form responses are 38 per cent. This is within normal limits but shows a tendency toward emotional constriction. In addition, form and shading responses (FK+F+Fc) constitute only 45 per cent of the record. This is low and detracts from the quality of control when tact and finesse are required in handling personal relationships.

Animal content is high (48 per cent), and the number of FM (animal movement) responses exceeds the number of M responses, showing some emotional immaturity and instability, especially since the CF (color-form) responses exceed the number of FC (form-color) responses, in a ratio of 1.62 : 0.62. This shows a tendency to respond more impulsively to affective stimulation than if the FC determinant dominated, in which case a more adaptive type of social response would be expected. Response to the colored cards is 36 per cent, but the ratio of M : sum C is 1.3 : 1.0, showing more interest in color than is demonstrated by effectiveness in its use.

13. Burgemeister, B. B., and Tallman, G.: Rorschach Patterns in Multiple Sclerosis, Rorschach Res. Exch. **9**:111-122 (Sept.) 1945.

Emotional control is within normal limits, although somewhat rigid. It is believed that the pattern in general indicates sufficient interference with optimal mental functioning and with productiveness to differentiate it in many ways from the records of well adjusted normal adults.

Space does not permit a detailed analysis of the Rorschach data, and we have included graphs illustrative only of our two extreme groups, i. e., the patients with idiopathic petit mal and those with traumatic epilepsy (charts 1 and 2).

In our group of patients with a combination of idiopathic petit mal and grand mal seizures, creative thought has less influence over infantile drives than in the group with petit mal seizures only. Less efficient emotional control and a greater degree of emotional constriction are also apparent. Although the response to social

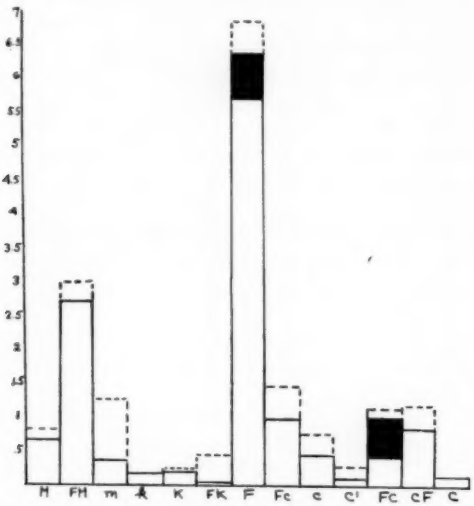

Chart 2.—Rorschach patterns in our group of adults with traumatic epilepsy.

In our group with traumatic epilepsy the total number of responses is lower (13.91, as compared with 15 for the petit mal patients). The number of M's is also more limited, being 0.63, in contrast to 2.0. The discrepancy between capacity and production (W : M) is much greater in this group, being 9 : 1, whereas it is 3.2 to 1 for our petit mal patients. Initial reaction time per card is the longest of those for any of our adult groups, although still fast (13.9 seconds).

Fewest popular responses occur here, and animal content is most overemphasized, with the M : FM ratio 0.63 : 2.68. This is a less satisfactory balance than that for any of our adult patients.

Emotional constriction and decreased flexibility of thought are shown by a form content of 58 per cent, combined with poorer form quality. There is a reversal in the ratio of M : C and (FM+m) : (Fc+c+C'), being 1 : 2.7 and 2 : 1, respectively. This shows less satisfactory adjustment and more conflict than if the relationship were not reversed.

Animal content is also higher (59 per cent) than in any of our groups, reflecting an even more naive intellectual approach and overemphasis on infantile drives as a basis for motivation.

Response to color is the highest of any of our groups (40.5 per cent). It is significant, however, that 50 per cent of the FC responses in the traumatic epilepsy records are of minus form quality. Our data indicate that these patients have the most difficulty of any of our groups in handling this determinant. Color naming appears in two records, and some of the records have other definite signs of organic damage.

On the whole, we believe that evidence exists for the most interference with mental efficiency on an organic basis in these patients and for less satisfactory adjustment than in any of our epileptic categories.

stimulation is less impulsive than it is among our petit mal patients, difficulty in maintaining consistent control is shown in this area. Slightly more interference with mental functioning in general is evident among this group than among patients of the petit mal type.

The records for the idiopathic grand mal group show that inner productivity and control are less adequate than in either the idiopathic petit mal or the petit mal and grand mal group. Even more emphasis is placed on infantile motivation, and difficulty is apparent in maintaining efficient social control. The latter is a feature frequently found among patients with diffuse organic damage to the brain, when flexibility of thought has decreased to a measurable degree.

In the group with symptomatic epilepsy the ratio of drive to capacity for productiveness is definitely out of balance, being three times as poor as that of the petit mal, the petit mal and grand mal or the grand mal group. It is more than five times that of the ideal ratio (2:1). A still more naïve intellectual approach and increased rigidity of control appear than in any of the groups with idiopathic epilepsy. Less efficiency in handling social contacts is indicated also. It is believed that evidence exists for much more interference with mental efficiency on an organic basis than is shown in any of the idiopathic categories and for a lowering in the level of functioning.

The Rorschach pattern of our group of patients classified as having traumatic epilepsy reveals the least productiveness and the greatest interference with logical thought (chart 2). Poorer social and emotional adjustment and shrinkage of the personality are much more apparent.

One of the most striking features of the pattern of our traumatic epilepsy group is the difference in form quality, as shown by the shaded area (chart 2), indicating poor (minus) form. This poor form level occurs more frequently in the group with traumatic epilepsy than in the groups with idiopathic petit mal and shows a relatively greater difficulty in perceiving concepts clearly, as well as difficulty in handling the colored cards.

Time does not permit a detailed discussion of all Rorschach determinants and their rationale. These will be presented in a subsequent publication. It seems worth while, however, to consider here one determinant, M, or human movement, because it correlates so well with our findings on the intelligence tests.

The ability to see human movement in the Rorschach cards depends entirely on the productivity of the subject, since the cards themselves are inanimate objects, or blots. It is agreed among Rorschach investigators that the M, or human movement, determinant measures creativeness and degree of imaginative thought better than any other determinant, even though its interpretation depends on the total gestalt. The M determinant is conspicuously absent in persons with defective intelligence and in patients with severe damage to the brain. In a person of average intelligence, such as our sample includes, Rorschach [4] stated that one or two M's represent a noncreative, predominantly reproductive intelligence, and that three to five M's represent more specifically a high average intelligence at an intelligence quotient level of 110. Other workers have confirmed this hypothesis.

In table 4 the relation between the intelligence quotient, as measured by the Wechsler-Bellevue Intelligence Scale, and the average number of M's in the Rorschach record is given according to our classification based on type of convulsions.

In all our epileptic patterns the number of M's is low, with the petit mal group (two M's) coming closest to the desired number for patients at this intellectual level. This finding agrees well with our intelligence test data showing average intelligence among our epileptic patients in general, as does the close relationship which exists between the intelligence test scores and the number of M's in the Rorschach records according to our various categories.

In addition to furnishing a good index of intelligence, the number of M's in the Rorschach responses also gives a guide to the richness of the inner life. Klopfer and Kelley [14] postulate that when a marked reduction in M's occurs, "the inner life of such a subject is not rich enough to give him the necessary poise and security for his dealings with the outer world." This is the type of evidence which may be elicited from a study of Rorschach protocols.

SUMMARY AND COMMENT

Since each type of clinical attack manifests itself in a particular form and degree of disturbance of conscious awareness, it occurred to us to study the intelligence and personality of epileptic children and adults in connection with a clinical etiological classification.

TABLE 4.—*Number, Intelligence Quotient and Average Number of M's in the Rorschach Responses of Our Adult Groups, According to Our Epilepsy Classification*

Classification	No.	I. Q.	Average No. of M's in Rorschach Material
Idiopathic petit mal	10	108.70	2.00
Idiopathic petit mal and grand mal	40	103.54	1.40
Idiopathic grand mal	74	98.50	1.16
Symptomatic	20	98.13	0.73
Traumatic	23	92.52	0.63

Our findings indicate that such an etiological classification based on commonly accepted clinical types does reveal a relationship of intelligence, personality and type of seizure among groups of epileptic patients.

Additional evidence regarding the relation of intellectual efficiency to epilepsy seems indicated in our observation that the intelligence quotient is higher if the onset of grand mal seizures appears late than if it appears early in the child's life.

Since learning depends on the transmission of sensations from the environment through the nerve impulses to be integrated into a state of conscious awareness, from which intelligence is derived, it would seem logical to expect that the later the onset of the process, the smaller the intellectual impairment should be.

Primarily as the result of the teachings of William A. White and Adolf Meyer, medicine in the past few years has understood that the organism must be treated as a whole. The acceptance of this dictum has resulted in what is now symbolized in the term psychosomatic medicine. While this term has served admirably in a pragmatic way, it leaves something to be desired in the sense that the term psyche in everyday clinical thinking seems to exclude a consideration of the intellect of the organism. This is probably because the academic psychologists have taken it to

14. Klopfer, B., and Kelley, D. M.: The Rorschach Technique, Yonkers-on-Hudson, N. Y., World Book Company, 1942.

their bosom in connection with education, so that it no longer appears to be a part of medicine, and also because medicine itself has displayed only a secondary interest in the intelligence of the organism.

However, there is considerable evidence that emotions and intelligence are inextricably entwined and that one cannot be considered without the other in psychic manifestations.

In 1945 Spitz [15] presented evidence which pointed to the possibility that depriving infants of normal maternal warmth and protection may actually cause retardation in mental growth, particularly in perception and locomotion.

In our own work with mentally retarded children,[16] we found that well adjusted children responded better to treatment than those who were not well adjusted, as indicated by the Rorschach test.

It was natural, therefore, that this study should include a simultaneous investigation of the emotional status of the individual epileptic patient and his intellectual status.

In the light of such an approach, consisting of a simultaneous investigation of the emotional and intellectual status of the individual epileptic patient in terms of the type of seizure, our data, we feel, clearly indicate a close correlation of these three factors and show fine shades of difference in intelligence and personality, even though the intelligence of the group as a whole falls at the average level. A lumping together of all types of epilepsy by previous investigators has served merely to obscure these finer differences. We therefore believe it is a mistake to combine the petit mal, grand and petit mal, and grand mal types of seizure under the heading of "idiopathic" when studying the results of intelligence and performance tests. Although our group of petit mal patients is small, the presence of the grand mal seizure, whether it be idiopathic or localized in nature, appears to have a more detrimental effect on the intelligence quotient than can be discerned when the petit mal type of seizure exists alone.

In any event, we feel it is significant that petit mal epilepsy, with its transitory lapses of consciousness, and therefore relatively mild clinical manifestations, should show the highest intelligence quotient level and the least amount of personality deviation, whereas the intelligence quotient becomes lower and the personality deviations are more marked as the types of seizures become severer.

The results of the Rorschach test for our adult group likewise reveal a reduction in the number of M's in all forms of epilepsy, even in the petit mal type. Since a reduction of M's is an indication of "organicness," as is quite obvious in the symptomatic and traumatic forms, it would appear that the Rorschach records of our patients show some degree of "organicness" in all forms of epilepsy, even in the so-called idiopathic types.

At this time we are not prepared to say why the average intelligence and performance quotients of our children with petit mal epilepsy, as well as the quotients for our adults with petit mal, are so far above the means for the other groups and for the general population. It is not clear from our data whether this difference may

15. Spitz, R. A.: Hospitalism, in Freud, A.; Hartmann, H., and Kris, E.: The Psychoanalytic Study of the Child, New York, International Universities Press, Inc., 1945, vol. 1, pp. 53-74.

16. Zimmerman, F. T., and Burgemeister, B. B.: The Effect of Glutamic Acid on Borderline and High-Grade Defective Intelligence, New York State J. Med. **50**:693-697, 1950.

be due to an error in sampling of our petit mal patients or whether our total distribution represents a cross section of the population with potentially higher intelligence than the quotients indicate. If the latter assumption is correct, our findings may be explained by the presence of disturbing physiological and psychological factors which interfere with optimal functioning. Our Rorschach records do show the influence of such interfering components, so that it seems probable this hypothesis may be valid. If so, our intelligence test results may be interpreted on a psychosomatic basis, in keeping with the nature of the epileptic disorder itself.

Finally, we feel that our data shed light on the controversial problem of the "epileptic personality." While no one definite pattern emerges which distinguishes the "epileptic personality" from the "nonepileptic personality" on the basis of data from the Rorschach and intelligence tests, differences in personality pattern do occur among various groups of epileptics when etiology is used as the criterion of differentiation. Our results seem to demonstrate some interference with mental functioning in all of our epileptic groups, and less satisfactory personality adjustment than might be found among similar samples of persons free from epileptic seizures. We are not prepared to say whether difficulty in adjustment stems from the presence of epilepsy itself, whether it represents a reaction to the convulsive seizure or whether it is a combination of physiological and psychological factors. We are convinced, however, from our findings that differences in degree of interference with mental functioning and with personality adjustment do exist among patients with epilepsy and that these may be detected in intelligence and personality tests.

CONCLUSIONS

Our findings indicate that a relationship exists between the degree of disturbance in conscious awareness and the adequacy of mental functioning in groups of epileptic patients.

An etiological classification based on commonly accepted clinical seizure types reveals differences in intelligence and personality in these groups of epileptic patients.

Petit mal epilepsy, with its transitory lapses of consciousness and relatively mild clinical manifestations, shows the highest intelligence quotient and the least amount of personality deviation among children and adults, whereas in the severer types of seizures the intelligence quotient is lowered and productiveness is more curtailed.

The intelligence quotient is higher among children if the onset of the grand mal seizure appears late than if it appears early in the child's life

Our Rorschach records seem to demonstrate some degree of "organicness," even among the idiopathic types, interference with mental functioning being most pronounced in our groups with symptomatic and traumatic epilepsy.

All these conclusions are made possible by a method of classification which utilizes finer degrees of differentiation than the older dichotomy of "organic-nonorganic" epilepsy.

PSYCHOLOGICAL EFFECTS OF CHRONIC BARBITURATE INTOXICATION

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THE PURPOSE of this paper is to present studies of the changes in performance in a number of psychological tests by human subjects who were experimentally addicted to barbiturates. The clinical, biochemical and physiological findings of this experiment have been reported by Isbell and associates.[1] These investigators used five former morphine addicts who volunteered for the experiment. The subjects received secobarbital sodium (seconal®), amobarbital sodium (amytal®) and pentobarbital sodium (nembutal®) orally for periods of 92 to 144 days. The doses employed were sufficiently large to induce mild to severe intoxication continuously. The results indicated that chronic barbiturism was similar to chronic alcoholism. The effects of the same dose of the drug varied greatly from day to day in the same subject, and striking differences were found between subjects. After abrupt withdrawal of barbiturates, definite abstinence phenomena—anxiety, anorexia, nausea and vomiting, convulsions of a grand mal type and psychosis clinically resembling alcoholic delirium tremens—developed. The psychosis was characterized by anxiety, agitation, insomnia, confusion, disorientation mainly for time and place but not for person, delusions and visual and auditory hallucinations. After recovery from chronic intoxication and withdrawal, the patients showed no signs of residual physical impairment.

We have been unable to find reports of psychological measurements taken during chronic administration of barbiturates, although a number of reports on the effects of single doses have appeared in the literature. Sargant and associates,[2] in a study of the effects of single doses of alcohol and amobarbital on intelligence scores (Cattel Tests IIA and IIB, tests of verbal intelligence), found that 0.065 to 0.195 Gm. of amobarbital caused an average drop in the intelligence quotient of 4.6 points. This loss in the intelligence quotient was slightly less than that obtained with 20 cc. of absolute alcohol. Sargant and his collaborators concluded that persons do not vary in susceptibility to the drug as might be expected. If a man's score on one of the Cattel tests was known, the score on the subsequent form could be better predicted when the amobarbital was given than when it was not.

From the Research Division, United States Public Health Hospital, Lexington, Ky. (National Institute of Mental Health), and the University of Kentucky.

1. Isbell, H.; Altschul, S.; Kornetsky, C. H.; Eisenman, A. J.; Flanary, H. G., and Fraser, H. F.: Chronic Barbiturate Intoxication: An Experimental Study, Arch. Neurol. & Psychiat. **64**:1-28 (July) 1950.

2. Sargant, W.; Slater, P.; Halstead, H., and Glen, M.: Effects of Alcohol and Sodium Amytal on Intelligence-Test Score, Lancet **1**:617-618, 1945.

Damrau [3] studied the effects of barbiturates on the intellectual performance of 50 neurotic patients, as measured by the Pinter General Ability Tests. The measurement of intellectual ability was made 30 minutes after the subjects had received 0.048 Gm. of pentobarbital sodium. The results revealed an increased performance level with the test used. Damrau concluded that anxiety was a disturbing influence in these patients, which prevented their functioning at a high level of efficiency, and that the improvement in ability obtained after the drug was due to a reduction of the anxiety.

Most of the authors who have investigated the effects of barbiturates on personality have been concerned with its use in narcosynthesis and narcotherapy. Layman [4] showed that schizophrenic patients exhibited some improvement on psychological test scores after intravenous injection of amobarbital. Thorner [5] concluded that pronounced mental changes may be produced in some psychotic patients, the action of the drug being largely of an anti-inhibitory nature. Kelley and his co-workers [6] obtained similar results on the Rorschach examination after injections of amobarbital. The responses were qualitatively less bizarre and less stereotyped, allowing finer nuances of personality interpretation. Sollmann,[7] summarizing the use of amobarbital in narcotherapy, stated:

> The inebriation grade of narcosis facilitates psychic control and renders the patient responsive to questioning, analogous to the "hypnotic state." This is utilized in the analysis of psychosis and in the treatment of schizophrenic patients.

EXPERIMENTAL MATERIAL AND METHODS

Subjects.—Five male volunteers who were former morphine addicts serving sentences for violation of the Harrison Narcotic Act were used in this experiment. All had been abstinent from morphine and other drugs for at least three months prior to this study. All had records of alcoholism, delinquency and vagrancy, and all had served more than one sentence for violation of the Narcotic Act. During previous sentences, all had at least one psychiatric examination, and an additional evaluation was obtained before the investigation was started. There was no personal or familial history of epilepsy or psychosis in any of the subjects.

On the basis of the psychiatric interview and the Rorschach examination, the following personality characterizations were made: S-1,[8] aged 42, a character disorder with anxiety, inadequacy and dependency; S-2, aged 42, a character disorder with compulsive features; P-3, aged 40, a character disorder with dependency, inadequacy and depressive tendencies; P-4, aged 48, a character disorder with inadequacy; A-5, aged 30, a character disorder with schizoid traits.

General Test Conditions.—The patients were constantly observed by trained attendants in a special ward devoted to clinical investigation. Two of the subjects received secobarbital; 2, pentobarbital, and 1, amobarbital. All drugs were administered orally in the form of 0.1 Gm. capsules or tablets.

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3. Damrau, F.: Psychometric Evaluation of Sedatives, M. Rec. **159**:349-351, 1946.

4. Layman, J.: A Quantitative Study of Certain Changes in Schizophrenic Patients Under the Influence of Sodium Amytal, J. Gen. Psychol. **22**:67-86, 1940.

5. Thorner, M. W.: The Psycho-Pharmacology of Sodium Amytal, J. Nerv. & Ment. Dis. **81**:161-167, 1935.

6. Kelley, D.; Levine, K.; Pemberton, W., and Lillian, K. K.: Intravenous Sodium Amytal Medication as an Aid to the Rorschach Method, Psychiatric Quart. **15**:68-73, 1941.

7. Sollmann, T.: A Manual of Pharmacology and Its Application to Therapeutics and Toxicology, Philadelphia, W. B. Saunders Company, 1948, p. 684.

8. The subjects are designated by a number with a letter prefix. The letter represents the drug taken by the subject: A, amobarbital; P, pentobarbital, and S, secobarbital.

Tests Used.—The following psychological procedures were employed: (1) the 1937 revision by Terman and Merrill[9] of the Stanford-Binet intelligence test, Forms L and M; (2) the Rorschach test, the criteria of Beck[10] being used for scoring, except for the use of FM (animal movement)[11]; (3) Kohs Block Design[12]; (4) Bender Gestalt test[13]; (5) Draw-a-Man test, the directions and criteria for interpreting the results being those set forth by Machover,[14] except that the subjects were instructed to put the drawings for both the male and the female on the same sheet of paper; (6) Digit-Symbol test, taken from the Wechsler-Bellevue test of adult intelligence,[15] modified by making six sets of alterations in the order of the symbols. Each time the Digit-Symbol test was administered, the subject received a different key until all six keys had been given. In further testing, the procedure was repeated. The modification was designed to lessen practice effects.

EXPERIMENTAL PROCEDURE

Preliminary.—During this period control tests were obtained. All tests were administered once, and the Bender Gestalt, Draw-a-Man and Digit-Symbol tests were given before and approximately every hour for five to six hours after the administration of single doses of the various barbiturates. This period lasted 14 to 21 days.

Chronic Drug Administration.—During this period the patients received large amounts of barbiturates daily. The drug used, the total amount taken, the number of days and the dosage schedule are summarized in table 1. The Digit-Symbol, Bender Gestalt and Draw-a-Man tests

TABLE 1.—*Dosage During Chronic Barbiturate Intoxication*

Patient	Drug	No. of Days on Drug	Dose, Gm. per Day During Chronic Administration *		Total Amount of Drug Taken, Gm.
			1st to 21st Day	22d Day to End of Period	
S-1	Secobarbital sodium	92	0.4-1.3	1.3-1.6	110.1
S-2	Secobarbital sodium	132	0.4-1.6	1.6-1.8	214.3
P-3	Pentobarbital sodium	119	0.4-1.3	1.3-1.8	175.3
P-4	Pentobarbital sodium	144	0.4-1.3	1.3	177.7
A-5	Amobarbital sodium	104	0.4-3.0	3.0-3.8	310.2

* The total daily amount was divided into five doses. At 5:00 a. m. a small "eye-opener" dose of 0.1 to 0.2 Gm. was given, and larger doses were administered at 9 a. m., 2 p. m., 7 p. m. and 11 p. m.

were administered before and 30 to 60 minutes after the 9 a. m. dose at intervals of three to seven days. The Kohs Block Design test and the Rorschach test were administered once before the 9 a. m. dose, on the eighteenth day of chronic intoxication.

Withdrawal Period.—Withdrawal of the barbiturates was abrupt and complete, except in the case of S-1, who had to be returned to administration of the drug during the ninth day of

9. Terman, L. M., and Merrill, M. A.: Measuring Intelligence: A Guide to the Administration of the New Revised Stanford-Binet Tests of Intelligence, Boston, Houghton Mifflin Company, 1937.

10. Beck, S. J.: Rorschach's Test: I. Basic Processes, New York, Grune & Stratton, Inc., 1944.

11. Klopfer, B., and Kelley, D. M.: The Rorschach Technique: A Manual for a Projective Method of Personality Diagnosis, Yonkers-on-Hudson, New York, World Book Company, 1942, p. 63.

12. Kohs, S. C.: Intelligence Measurement: A Psychological and Statistical Study Based upon the Block-Design Tests, New York, The Macmillan Company, 1923.

13. Bender, L.: A Visual Motor Gestalt Test and Its Clinical Use, Research Monograph 3, American Orthopsychiatric Association, Menasha, Wis., George Banta Publishing Company, 1938.

14. Machover, K.: Personality Projection in the Drawings of the Human Figure: A Method of Personality Investigation, Springfield, Ill., Charles C Thomas, Publisher, 1949.

15. Wechsler, D.: The Measurement of Adult Intelligence, ed. 3, Baltimore, Williams & Wilkins Company, 1944, p. 185.

abstinence because of the imminence of physical collapse. The withdrawal period covered 12 to 13 days for the remaining four subjects. The Bender Gestalt, Digit-Symbol and Draw-a-Man tests were administered every two or three days until the patients reached levels equal to those obtained during the preliminary period. The Rorschach test was administered once or twice during withdrawal, either on the day following convulsions or during a psychotic episode.

Recovery Period.—During this period, which covered two to three months, all tests were administered to all subjects at least a month after withdrawal of the barbiturates.

Reintoxication.—After the recovery period was completed, four of the subjects were abruptly placed on the same dose of barbiturates they had been receiving immediately before withdrawal began. The Digit-Symbol, Bender Gestalt and Draw-a-Man tests were administered once before and approximately 60 minutes after the 9 a. m. dose. This period lasted three or four days.

TABLE 2.—*Scores in Stanford-Binet Intelligence Tests in Preliminary and Recovery Periods*

Subject	Intelligence Quotient	
	Preliminary Period (Form L)	Recovery Period (Form M)
S-1	87	88
S-2	93	111
P-3	90	106
P-4	81	82
A-5	114	119

TABLE 3.—*Kohs Block Test Scores in Various Periods of the Experiment*

Subject	Preliminary Period		Period of Chronic Intoxication		Period of Recovery	
	Raw Score	Mental Age *	Raw Score	Mental Age *	Raw Score	Mental Age
S-1	29	10-4	20	9-3	29	10-4
S-2	92	15-2	108	16-1	110	16-9
P-3	60	12-11	60	12-11	87	14-10
P-4	32	10-8	25	9-11	37	11-1
A-5	52	12-4	47	11-11	75	14-0

* The first figure in the "Mental Age" columns refers to years; the second, to months.

RESULTS

Stanford-Binet Test.—Scores on this test are shown in table 2. Since this test was used mainly as a measure of possible residual intellectual impairment, it was administered only during the preliminary and recovery periods of the experiment. All the subjects showed an increase in total intelligence quotient points on the second administration. The large increase in two of the subjects, S-2 and P-3, and the slight increases in the other three subjects, probably reflect increased rapport between the subjects and the examiner and the patient's increased familiarity with the testing.

Kohs Block Design Test.—Table 3 summarizes the results obtained with the Kohs Block test. Three of the five subjects, S-1, P-4 and A-5, showed a decrease in performance during chronic drug administration which resulted from inability to do the task as quickly and with the same number of moves. Their ability to perceive the relationships remained unimpaired, for they were able to complete the same number of designs when allowed to exceed the time limit. S-2 and P-3 showed no deterioration in their performances, and clinically these two subjects did not exhibit as

pronounced signs of intoxication as did the other subjects. During the recovery period, all subjects reached a level of achievement as good as, or better than, that attained in the preliminary period.

Rorschach Test.—Table 4 summarizes the results of the Rorschach test for each subject. The follow-up summary of the Rorschach test is not given for the findings approximated, both qualitatively and quantitatively, those of the Rorschach test administered during the preliminary period.

The determinant that appeared to be altered in most of the subjects during chronic intoxication was a decrease in the percentage of F responses, which was observed in all subjects except A-5. Other changes that occurred during chronic intoxication reflected individual differences rather than any common characteristics of behavior. However, the possibility exists that personality changes greater than those found in the Rorschach test were present but were masked by familiarity with the test. The fact that the test had to be administered in the morning when the

TABLE 4.—*Rorschach Results for Individual Subjects*

Scoring Category	S-1 Administrations				S-2 Administrations			P-3 Administrations			P-4 Administrations			A-5 Administrations		
	1	2	3A	3B*	1	2	3*	1	2	3	1	2	3*	1	2	3
W	2	3	1	0	2	2	2	10	12	9	3	3	2	4	2	1
D	12	15	17	30	11	10	8	1	2	1	28	25	20	18	20	14
Dd	2	0	2	14	18	9	0	0	0	0	1	1	1	0	0	0
R	16	18	20	44	31	21	10	11	14	10	32	29	23	22	22	15
Z	3.5	4.5	1.0	3.0	2.0	5.0	1.0	30.0	36.5	16.5	15.5	8.0	1.0	17.5	16.5	12.5
M	0	1	0	2	0	0	1	0	1	0	0	0	0	6	5	4
FM	0	1	2	7	3	4	0	0	1	0	2	1	0	1	2	1
C	0	0	0	0	0	0	0	4.0	3.5	6.5	1.5	.5	1.0	1.5	.5	.5
Y & V	0	2	0	0	0	1	1	0	1	2	3	1	2	6	5	4
F+%	81	73	65	53	90	88	63	50	45	66	85	67	53	50	58	57
A%	69	72	65	71	65	52	78	54	57	80	63	62	78	55	55	47
P	5	7	3	3	2	2	2	2	3	1	3	2	1	6	5	5
T/1R (sec.)	38	13	10	12	43	19	53	37	9	6	18	17	11	32	20	58
T/R (sec.)	61	45	21	..	49	45	..	74	46	20	36	24	..	59	39	73

* Rorschach examinations during withdrawal psychosis. Administrations are numbered consecutively: 1, during the preliminary period; 2, during addiction; 3, during withdrawal. The test was administered to S-1 twice during withdrawal. All scoring categories are those of Beck,[10] with the exception of FM, taken from Klopfer and Kelley.[11]

degree of intoxication was minimal may also have tended to minimize changes in the Rorschach results.

Rorschach tests obtained during withdrawal psychoses in subjects S-1, S-2 and P-4 revealed the following changes: 1. The form level (F%) dropped somewhat from the normal. 2. There was a tendency for the stereotypy, as represented by the animal per cent (A%) to increase and for the number of popular percepts to decrease. 3. Although the subjects were hallucinating continually, a rise in fantasy was found only in S-1. 4. Inappropriate conduct with the cards was found; e. g., the subject might put them under a pillow. 5. Bizarre and contaminated responses were obtained. 6. The subjects often did not remember what they had perceived immediately after reporting it.

A-5, who did not become psychotic during abstinence, showed a decided slowing. The number of percepts decreased, and the time to first response (T/1R) and time per response (TR) were approximately twice the values obtained on the previous administration. The slowing may have been due to postconvulsive confusion, since A-5 had three grand mal seizures on the day before the test was administered. P-3, whose record was speeded up, had only one convulsion.

Digit-Symbol Test.—During the single dose tests, all the subjects showed a decrease in efficiency one-half to one hour after the drug was administered. Five to six hours after the drug was given, their scores approximated those of the control tests.

Figure 1 shows the achievement level for each subject during the course of chronic intoxication. The patients, with one exception (S-2), showed a quick decline in their ability to do the Digit-Symbol tests. The low point in efficiency was reached within 30 days after the start of chronic intoxication. This was followed by a gradual increase in efficiency, the peak being reached 30 to 70 days after the start of chronic intoxication. Their efficiency then tended to drop slightly,

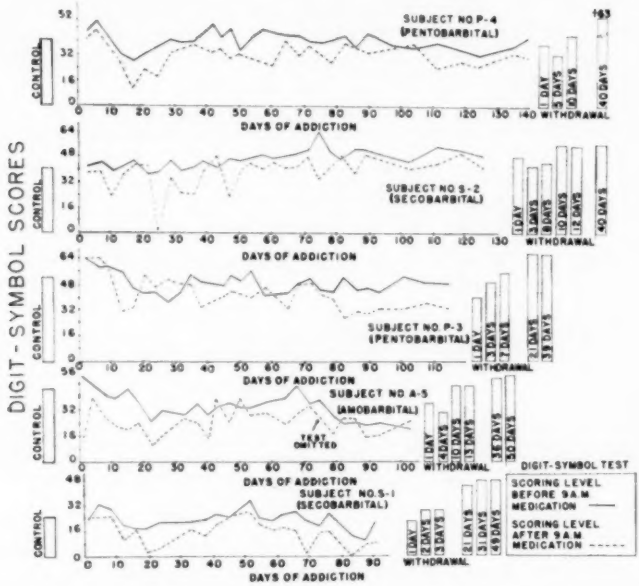

Fig. 1.—Results for individual subjects in the Digit-Symbol test. Scores represent the total number of symbols completed correctly in 90 seconds. Control scores are the means of all tests administered before addiction began (excluding those after single doses of barbiturates).

and the decrease was especially marked if the dose had been increased. With a few exceptions, postmedication scores (after the 9 a. m. dose) were always less than the premedication scores. S-2, who continually increased his efficiency during chronic intoxication, exhibited the lowest grade of intoxication, as judged by clinical criteria. The gradual increase might be attributed to this subject's pronounced compulsiveness.

Table 5 shows the means (M) and standard deviations (S.D.) of the premedication and the postmedication digit-symbol scores for each subject. The mean was lower for the postmedication administrations, and the SD was higher for all subjects with the exception of S-1. S-1's lower standard deviation occurred because the mean level of achievement after medication in this subject was so low

that a high standard deviation would be impossible. Inspection of the standard deviations and of figure 1 reveal that scores after medication fluctuated more than the scores before medication. Low scores after medication were often empirically correlated with food intake—impairment was greater if the subject had not eaten a full meal prior to medication.

After completion of the experiment, a control study was conducted with the Digit-Symbol test for a period of six weeks using five different subjects. The procedure was the same as that employed during chronic intoxication except that no drugs were given. No significant differences were found in the scores of the first and second administrations on the same day. The fluctuations were not as great as those obtained under conditions of chronic intoxication. Thus, the fluctuations and the consistently poorer achievement on the postmedication tests than on the premedication tests during addiction were the result of the action of the drug and not of the experimental design.

Draw-a-Man Test.—Changes in the drawings that occurred after single doses of barbiturates during the preliminary period, although of the same type, were not as great as those observed during chronic intoxication.

TABLE 5.—*Means and Standard Deviations of the Premedication and Postmedication Scores of the Digit-Symbol Test*

Subject	Premedication Score		Postmedication Score	
	M	S. D.	M	S. D.
S-1	23.4	16.9	15.8	8.7
S-2	47.3	5.8	39.0	10.2
P-3	49.1	6.1	41.8	9.9
P-4	41.1	5.3	32.5	10.5
A-5	34.7	8.0	25.5	8.1

Figure 2 *A* shows A-5's first representation of the human form. During the single dose test the male figure lost his clothes and, finally, his hat. During chronic intoxication, the female was drawn with more hair than the male, but in periods of great intoxication the hair of the male resembled that of the female. The shoulders of the male were exaggerated, but the hips became very feminine and resembled those of the female. These phenomena tended to progress as the experiment continued, the feminine emphasis on the hips of the male becoming more pronounced, as shown in figure 2 *B*. Despite the fact that A-5 prided himself on his virility and his success with women, his drawings indicated a release from control of repressed homosexual factors, and the exaggeration of the shoulders of the male suggested an infantile attempt to show masculinity.

C and *D* of figure 2 show the first drawing made by S-2 during the preliminary period and the one done on the fifty-fifth day of intoxication. As the experiment progressed, S-2 drew the male and female figures in greater and greater detail. He erased a great deal and took more time to complete the drawings. The trouser fly on the male figure was drawn conspicuously, and the hands were hidden in the trouser pockets. The feet and fingers of the female figure were of phallic form. The drawings indicated an increase in compulsive features in the personality of S-2, a masculine projection on the female and projection of masturbatory guilt (prominent trouser fly and hiding of hands). It is of interest that this subject masturbated during the psychosis following withdrawal of barbiturates.

In the early drawings of P-4, the hair of the female figure was very disorderly, whereas the hair of the male was neatly arranged and parted in the middle. As chronic intoxication progressed, the hair of the female became less disarranged and that of the male more untidy. These changes may possibly indicate that under the effects of pentobarbital P-4's feelings of masculine inadequacy were dissipated.

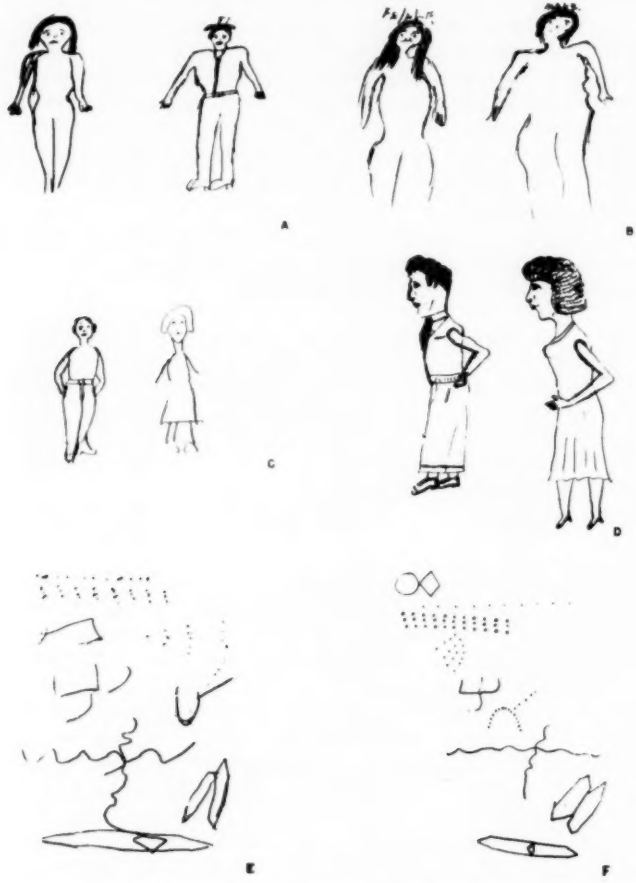

Fig. 2.—Representative drawings made during different periods of the experiment and Bender Gestalt tests done by two subjects at approximately the same period of chronic intoxication.

The drawings of P-3 and S-1 did not change greatly during chronic intoxication. P-3 drew a "peanut man" representation of the human form throughout the experiment. As addiction continued, he tended to make the figures smaller and to put them in the upper left hand corner of the paper. The "peanut man'" representation was probably an attempt at evasion, and the small figures which were pushed into

the corner of the paper suggested that more constricted and depressive elements in his personality were being projected. The preliminary drawings of S-1 were characterized by fuzzy broken lines and with no differentiation between the male and female. During chronic intoxication, his pictures became more blurred and broken up than they were during the preliminary period. These drawings suggested a schizoid, inebriate, hysterical personality. No other change of significance occurred.

It was impossible to obtain drawings from S-1, S-2 and P-4 while they were psychotic after withdrawal of barbiturates. Ten to 20 days after medication was discontinued, and after the psychosis abated, the drawings were similar in quality to those obtained during the preliminary period.

Bender Gestalt Test.—In the preliminary period, regressions in the gestalt forms similar to those described in the following paragraph were observed an hour after large single doses. Recovery was rapid, coinciding in time with recovery of efficiency in doing the digit-symbol test.

During chronic intoxication the characteristic changes in the Gestalten included dots becoming circles, disturbances in the relationships of parts to wholes and figures to background and increase in the relative sizes of the figures. Subjects continually erased and marked over lines. S-1 occasionally inverted figure 5 (fig. 2 *E*). He would be aware of this inversion but, after two or three attempts to correct it, would give up and leave the figure inverted. There are some indications that all of the characteristic changes caused by the drugs were not simple regressions; for example, P-3 originally drew loops to represent dots; during chronic intoxication his loops tended to become dots (fig. 2 *F*), a more mature type of representation. During periods in which the degree of intoxication decreased, the Gestalt forms reverted to their original, or to more advanced, states. Individual differences between the subjects were very large. Some showed great impairment in performing this sensory-motor task, while others did not (fig. 2 *E* and 1 *E*). S-2, who exhibited very little deterioration in his premedication gestalt forms, did show a change in his postmedication forms.

Reintoxication.—During this period, four of the five subjects were abruptly placed on the same daily dose of barbiturates they had gradually attained during chronic intoxication. After 24 hours the performance of the subjects on the Draw-a-Man, Digit-Symbol and Bender Gestalt tests were lower than at any time during addiction.

COMMENT

The differences in the results of the premedication and postmedication administrations of the routine tests were greatest in the Digit-Symbol, less in the Bender Gestalt and least in the Draw-a-Man test. Apparently, tasks involving a time element are impaired most by barbiturates; those involving copying are disturbed less, and those requiring production of something stabilized in the past experience of the subject are affected least.

Marked differences in the changes observed in all the routine tests occurred in the same subject from day to day, even though the same dose of the drug was given. These results are in contrast with those of Sargant and associates.[2]

The Draw-a-Man test revealed very little basic change in personality during chronic intoxication. Nevertheless, there was some loss of the ego control, which was manifested by a greater magnitude of pathological personality projection. Id factors in the personality were expressed more easily by the subjects. This tended to become more pronounced as intoxication continued.

During chronic intoxication the Gestalten produced by the subjects tended to revert to a more primitive developmental level. However, some of the changes exhibited were not simple regressions, but were forms more advanced in the developmental scale. There was a disorientation of wholes to the background and of parts to wholes, which corresponded to the disorientation of the subject to his environment. This may be interpreted as a loss of conscious control or ego strength, allowing the release of basic personality demands. The same type of change was observed in the Rorschach test. Thus, reduction of ego control, with regression to a more primitive developmental level, seemed to be the most characteristic effect of the barbiturates on personality.

One may ask why only three of the five subjects became grossly psychotic during abstinence from barbiturates. No clues were found in neurological, physiological or biochemical studies. Results of the preliminary Rorschach examination suggest one possible answer. Differences were found in three determinants: There were a high percentage of F + responses and a lack of affect and fantasy in the Rorschach tests of the subjects (S-1, S-2, P-4) who became grossly psychotic during withdrawal, as opposed to a low percentage of F + responses and the presence of fantasy or affective responsiveness in the subjects who had a mild (P-3) or no (A-5) psychosis during withdrawal. Clinically, these two subjects were the ones who had the greater "pathological" personality manifestations before addiction began. Although the number of subjects was too small for any definite conclusions, this finding permits speculation as to the causes of psychotic behavior during withdrawal. Under extreme conditions of stress, the subjects who became psychotic had only one defense mechanism, that of repression. This one mechanism was not adequate for controlling the amount of stress present during the withdrawal period; hence a psychotic episode resulted. Affective expression or fantasy in the other subjects, although possibly pathological, allowed a release of tension and prevented the appearance of major clinical signs of psychosis.

It is also possible that the type of drug administered may have influenced the appearance and manifestations of the withdrawal psychoses. Although convulsions and a delirium have been described as following withdrawal of all the commonly used barbiturates, both psychosis and convulsions are known to occur more frequently after withdrawal of cyclobarbital than after withdrawal of phenobarbital or barbital.[16] A-5, the only subject who completely escaped the withdrawal psychosis, was the one patient who received amobarbital. Further work will have to be done to elucidate these problems.

As judged by the results obtained with Kohs Block, Stanford-Binet and Rorschach tests, as well as the results obtained by clinical and biochemical examinations, no permanent physical damage to the organism appears to result from barbiturate addiction per se when the barbiturate is administered under strictly controlled conditions, as in this experiment.

16. Pohlisch, K., and Panse, F.: Schlafmittelmissbrauch, Leipzig, Georg Thieme, 1934.

SUMMARY AND CONCLUSIONS

1. Five former morphine addicts, who volunteered for the experiment, were given sufficiently large doses of secobarbital, pentobarbital or amobarbital to induce continuous mild to severe intoxication for periods varying from 92 to 144 days. Six tests were used in an attempt to measure psychological changes during intoxication with abrupt withdrawal from the barbiturates. Three of the tests were administered routinely, the Digit-Symbol, the Bender Gestalt and the Draw-a-Man test. Three of the tests, the Rorschach, Stanford-Binet and Kohs Block tests, were administered only during specified periods of the study.

2. During the chronic intoxication period there was a quick decline in ability, followed by an increase in efficiency, which became maximal 30 to 70 days after the start of continuous medication. In only one instance was a score attained which was higher than control scores obtained after recovery from chronic intoxication. One of the subjects showed gradually increased efficiency on the Digit-Symbol tests from the very onset of chronic drug administration.

3. Oral administration of secobarbital, pentobarbital and amobarbital produced greatest impairment in performance of tasks involving speed, less impairment in tasks involving copying and least in tasks requiring production of behavior that had been stabilized in the past experience of the subject.

4. There were quantitative and qualitative differences in the effects of barbiturates on different subjects, and the same dose of a barbiturate affected the same subject differently on different days.

5. During chronic barbiturate intoxication, there was a partial loss of ego control, which was manifested by a greater magnitude of pathological personality projection in the projective techniques used. This trend became more pronounced as intoxication continued.

6. When the administration of barbiturates was discontinued, the performance of the subjects quickly reverted to the preaddiction level. No evidence of residual physical damage could be detected.

7. The results of Rorschach examinations obtained during the withdrawal from barbiturates, while the patients were psychotic, were very different from those obtained during the other periods of the experiment. The form level was comparatively low; bizarre responses were present; stereotypy (A %) increased, and the number of popular responses decreased.

8. It was observed that the preaddiction Rorschach patterns of the three subjects in whom major psychoses developed after abrupt withdrawal of barbiturates were characterized by high percentage of F + responses and deficiency in affect and fantasy. In the other two patients, the percentage of F + responses was low, and percepts related to affect or fantasy were present. The possible relations of these findings is discussed with reference to the susceptibility of certain personality types to the development of psychoses in reaction to the stress of barbiturate withdrawal.

STUDIES ON HEADACHE

Mechanism of Headache and Observations on Other Effects Induced by Distention of Bladder and Rectum in Subjects with Spinal Cord Injuries

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HEAD AND RIDDOCH,[1] in their classic experiments on patients with gross spinal cord injuries, found facilitation of spinal reflexes in that part of the cord below the level of the lesion and without suprasegmental control. It was shown that a sufficient stimulus to any receptive surface whose afferent fibers entered the distal stump of the cord was liable to evoke a massive response which overflowed widely into regions of the spinal cord normally associated with other reflexes (assuming that recovery from spinal cord "shock" and resumption of reflex activity of the distal stump of the spinal cord was not prevented by such complications as general infection and toxemia).

Especially investigated were abnormal autonomic reflex effects due to afferent impulses from bladder and bowel distention, both those of spontaneous occurrence due to urinary obstruction or flatulence and those artificially induced by inflation of the bladder or enemas. Outbursts of intense and profuse sweating, especially of the head and neck, and sensations of "fulness" in the head were described as manifestations of such bladder or bowel distention. Though other precipitating stimuli evoked bursts of sweating, abnormal conditions of tension in the bladder were the most frequent and potent cause of "spontaneous" hyperhidrosis in these patients with transverse lesions of the spinal cord.

Analysis of the distribution of reflex sweating in relation to the level of the lesion showed a correlation with Langley's reported levels of sympathetic outflow from thoracolumbar segments to the upper parts of the body. When the lesion was high enough that the upper thoracic segments were included in the distal stump of the cord, profuse sweating occurred in the head and neck, which derive their sympathetic innervation from the upper thoracic segments. Somewhat lower lesions (down to the sixth thoracic segment) resulted in reflex sweating only of the arms and upper portion of the trunk, since the impulses for the head and neck arising from levels of the cord above the lesion were no longer reflexly activated by afferent

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1. Head, H., and Riddoch, G.: The Automatic Bladder, Excessive Sweating, and Some Other Reflex Conditions in Gross Injuries of the Spinal Cord, Brain **40** (pts. 2 and 3):188, 1917.

impulses from the bladder. It was concluded that excessive sweating due to bladder distention (even in the head and neck) represented the activity of the nervous system below the level of the lesion of the spinal cord.

Since these classic studies, numerous other investigations of reflex autonomic responses occurring in spinal man have been carried out. The most recent detailed report of these phenomena, by Guttmann and Whitteridge,[2] included further analysis of reflex sweating and observations on other effects derived from bladder distention, such as vasoconstriction and diminished skin temperature in the extremities, flushing of the neck and face, nasal congestion, headache, hypertension and bradycardia. Though vasoconstriction of the toes and fingers and a very large rise in blood pressure occurred during bladder distention when the complete lesion was at or above the fifth thoracic level, these investigators emphasized that in the neck, face and nasal mucosa vasodilatation occurred. The seeming paradox of vasodilatation in the face and neck, on the one hand, concomitant with sweating in the face and neck and with vasoconstriction and sweating in the upper limbs, on the other (the last-mentioned responses all being manifestations of sympathetic activity in these parts), was explained on the basis of an adaptive mechanism. Flushing of the head and the nasal congestion were interpreted as due to passive dilatation of vessels secondary to increased blood pressure. Headache was believed to be related to a sudden rise in intracranial blood flow. Since it was shown that arteries of the head did not participate in the vasoconstriction occurring in the extremities and visceral bed, it may be assumed that they were passively dilated by the heightened intravascular tension brought about by vasoconstriction elsewhere.

Thompson and Witham,[3] in their clinical observations on patients with complete lesions of the cord, have also described the occurrence of spontaneous paroxysms of hypertension and headache. They confirmed the observation of reflex autonomic discharges due to bladder obstruction in cases of high lesions of the cord and demonstrated the abolition or prevention of these reflex effects by the injection of tetraethylammonium chloride. They emphasized the clinical importance of the vasomotor and sudomotor reflexes as a source of intermittent symptoms (sweating, headache) in paraplegic patients and discussed methods of management to prevent their occurrence.

PURPOSE OF INVESTIGATION

It was the primary purpose of this study to confirm the occurrence of headache during bladder or rectal distention in patients with high transverse lesions of the cord and to investigate the mechanism of such head pain. It was realized that such an investigation would also provide an opportunity to observe and measure the phenomena other than headache previously reported in association with bladder and rectal distention and the effects of various manipulative procedures on these. In this paper, it is not intended to analyze in detail all the experimental data obtained, especially those having no immediate bearing on the analysis of the mechanism of induced headache. A consideration of such data is being reserved for a subsequent communication, although certain aspects will be alluded to.

2. Guttmann, L., and Whitteridge, D.: Effects of Bladder Distention on Autonomic Mechanisms After Spinal Cord Injuries, Brain **70**:361, 1947.

3. Thompson, C. E., and Witham, A. C.: Paroxysmal Hypertension in Spinal-Cord Injuries, New England J. Med. **239**:291, 1948.

It has been shown that the largest proportion of clinical varieties of headache is due to distention and dilatation of relaxed cranial arteries.[4] Examples of such headache are migraine, the headache associated with essential hypertension and the headache associated with fever. Headache artificially induced by the intravenous injection of histamine is the classic experimental prototype of "vascular" headache. (This is not to be confused with the alleged clinical "histamine cephalgia," which to date has not been satisfactorily substantiated as an entity and which is probably a variant of migraine.) In contrast to migraine headache, in which both extracranial and intracranial arteries are painfully distended, the pain of experimentally induced histamine headache is derived almost exclusively from the distention of the intracranial pial arteries. It has been shown that such experimentally induced histamine headache is capable of complete elimination by means of artificially raising the intracranial pressure, thereby suppressing the pulsations of the painfully distended intracranial arteries.[5] An old clinical observation, and one which has been duplicated in the laboratory, is the transient reduction or abolition of the pain of migraine headache by compression of the carotid artery in the neck. It was thought worth while, therefore, to employ such manipulations in the study of "bladder" headache, especially in view of the hypothetical mechanisms proposed by Guttmann and Whitteridge.

METHODS

Bladder Distention Experiments.—Bladder filling was carried out, in stages and at a variable rate of flow, through a Foley indwelling catheter from a reservoir of sterile saline solution, elevated 100 cm. above the symphysis pubis and attached to the catheter by an intervening rubber tube with Murphy drip container and clamp. Frequent observations on the amount of fluid introduced into the bladder were recorded. In the first subject only, a calibrated water manometer was used to determine bladder pressure. In all subsequent experiments a Lewis recording cystometer, attached by a T tube to the catheter, provided a continuous ink-recorded graph of changes in intravesical pressure correlated with time. In some subjects, bladder contractions caused leakage of fluid around the catheter. These occurrences were recorded, and the amount of leakage was estimated. Usually it was possible to control leakage by slight traction on the catheter, bringing the Foley bag into closer apposition to the bladder neck.

Rectal Distention Experiments.—A large (no. 26) Foley catheter with a distensible rubber retention bag was inserted into the rectum. Tap water was introduced into the bag in 50 cc. increments by means of a large syringe. In the earlier experiments, no method of pressure recording was employed. The criterion for the degree of rectal distention consisted of the volume of fluid injected into the retention bag. In subsequent experiments, the bag was connected by a T tube and rubber tubing to the Lewis recording cystometer, permitting continuous pressure records.

Manipulative Procedures Designed to Modify Headache.—Elevation of Cerebrospinal Pressure: The effect on induced headache of experimentally increasing the subarachnoid pressure was studied. The apparatus and techniques for this procedure were set up prior to the beginning of bladder inflation. Spinal puncture was done with the patient in the lateral-recumbent position. Initially, bilateral compression of the jugular vein was carried out to ascertain the presence of free communication between the intracranial and spinal subarachnoid spaces and the manometer. After headache was induced by bladder distention, the cerebrospinal fluid pressure was

4. Wolff, H. G.: Headache and Other Head Pain, New York, Oxford University Press, 1947.

5. Schumacher, G. A., and Wolff, H. G.: Experimental Studies on Headache: A. Contrast of Histamine Headache with the Headache of Migraine and That Associated with Hypertension; B. Contrast of Vascular Mechanisms in Preheadache and Headache Phenomena of Migraine, Arch. Neurol. & Psychiat. **45**:199 (Feb.) 1941.

TABLE 1.—*Summary of Data Derived from Experimental Bladder Distention in Cases of Transverse Myelopathy*

Case No.	Patient	Age	Resting Blood Pressure	Level of Cord Lesion	Induced Bladder Changes		Associated Effects			Results of Manipulative Procedures			
					Maximum Filling (Vol. in Cc.)	Intravesical Pressure (Mm. Hg)	General	Blood Pressure Changes (Maximal Rise)	Headache Intensity (1+ to 10+)	Effect of Elevating Cerebrospinal Fluid Pressure	Effect of Tetraethylammonium Chloride (100 Mg. I. V.)		Effect of Carotid Compression
											On B. P.	On Headache	
1	J. P.	21	90/60	C5	200± (leakage around catheter)	Variable brief contractions to 50 mm.; slow fluctuations; average sustained 40-50 mm.	Sweating; pilomotor; "full chest"; spasms	220/115	10+	Headache abolished	.....	.....	.....
2	J. H.	22	110/80	C6	200	Steady; sharp rise initially; sustained at 60 mm.	Sweating; pilomotor; nausea	215/195	9+	Headache abolished	.....	.....	.....
3	G. C.	23	100/65	C6	250+ (leakage)	Variable; frequent strong contractions to 80-90 mm. throughout; interval pressures 25-40 mm.	Sweating; pilomotor; chills	230/150	6+	.....	.....	.....	.....
4	(a) E. K.	29	110/68	C8	450	Variable; slow, steady rise to 10-15 mm.; sustained with slow fluctuations	Sweating; pilomotor	205/115	10+	Headache abolished	Fall to 140/80	Abolished	.....
	(b) E. K.	29	118/75	C8	650	Variable; slow fluctuations to 10 mm. early; late rise to 15-20 mm.; sustained at 20 mm.	Sweating; pilomotor	225/128	10+	.....	.....	.....	Headache abolished in each of 3 trials
5	J. W.	31	120/90	T4	320	Variable; strong contractions to 80 mm. early; later sustained at 65 mm.	Sweating; pilomotor; "full throat"; chills; nasal congestion	205/120	9+	No effect (intraspinal block)	Fall to 152/90	Decreased to 6+	7 Trials: headache abolished twice; headache improved three times; headache unchanged twice
6	N. S.	29	100/70	T5	375	Steady; slow rise; sustained at 65 mm.	Sweating; pilomotor; "full throat"; chest pain	188/120	10+	No effect (intraspinal block)	.....	.....	.....
7	V. K.	45	105/85	T5	650± (leakage)	Variable; small contractions 30-40 mm.; later slow rise; sustained at 70 mm.	Pilomotor; abdominal pain	140/110	5+	.....	.....	.....	.....
8	J. L.	26	130/70	T7	450	Variable; powerful contractions to 105 mm. early; sustained at 70 mm.; slow fall; sustained at 40 mm.	Sweating	210/155	10+	Headache abolished	Fall to 155/112	Abolished	.....
9	J. D.	33	130/100	T8	475	Variable; mild contractions between 60 and 80 mm. early; sustained at 75 mm. with frequent brief contractions	Spasms	160/140	....	.....	.....	.....	.....
10	B. T.	26	125/80	T10	200± (leakage)	Variable; irregular contractions to 85 mm. early; sustained at 75 mm. late	None	130/90	....	.....	.....	.....	.....

increased by connecting with the lumbar subarachnoid space a flask of sterile fluid elevated 800 to 1,000 mm. above the spinal canal. The spinal needle and reservoir were connected by a three way stopcock and an intervening spinal fluid manometer. A clamp was released from the connecting tube when it was desired to increase the cerebrospinal fluid pressure rapidly and reapplied when further increase in pressure was no longer desired. To reduce pressure rapidly, the clamped tube was disconnected and the fluid allowed to flow freely from the subarachnoid space. Pressure readings were taken at intervals by means of the glass manometer.

Digital Compression of the Neck: The effect on the induced headache of bilateral digital compression over the area of maximum pulsation in the upper part of the neck was studied. Pressure was applied to the point of complete obliteration of palpable pulsations. A 10 to 20 second period of compression was maintained. Control observations on the effects of digital compression on the posterior part of the neck and mastoid bones were carried out.

Intravenous Injection of Tetraethylammonium Chloride: In subjects experiencing maximum effects from bladder distention, including marked hypertension and severe headache, tetraethylammonium chloride (etamon® chloride) was injected intravenously in doses of 100 to 300 mg.

TABLE 2.—*Summary of Data Derived from Experimental Rectal Distention in Four Cases of Transverse Myelopathy*

					Induced Rectal Changes					Results of Manipulative Procedures		
							Associated Effects			Effect of Tetraethylammonium Chloride (300 Mg. I.V.)		
Case No.	Patient	Age	Blood Pressure	Level of Cord Lesion	Rectal Bag Filling, Cc. Water	Intrarectal Bag Pressure, Mm. Hg	General	Maximum Blood Pressure	Headache Intensity (1-10+)	Blood Pressure	Headache	Effect of Carotid Artery Compression
1	J. P.	21	100/65	C5	600	114	Sweating, pilomotor, flush, spasms, chest fulness	180/104	5+	Fall to 114/76	Abolished	.....
2	J. H.	22	105/80	C6	350	130	Sweating, pilomotor, spasms	160/106	None	.....	.....	.....
3	E. K.	29	120/75	C8	575	...	Sweating, pilomotor, flush, spasms, chest fulness	180/105	9+	.....	.....	Headache abolished in each of two trials
4	B. T.	26	125/80	T10	700	...	None	140/100	None	.....	.....	.....

Observations on sweating, pulse, blood pressure, headache intensity, bladder pressure and cerebrospinal fluid pressure, already in progress prior to the injection, were continued.

RESULTS

Ten patients were investigated. They fell into three categories, depending on the site of the complete lesion of the cord: Category 1: lesion at or above the eighth cervical level, four patients; category 2: lesion between the first and seventh thoracic segments, inclusive, four patients; category 3: lesion between the eighth and twelfth thoracic segments, inclusive, two patients. All 10 patients had bladder distention experiments. On four of these, in addition, rectal distention experiments were carried out (tables 1 and 2).

In eight patients, all with lesions at or above the seventh thoracic level, bladder distention resulted in sweating, pilomotor reaction, slow pulse, significant hypertension and severe headache. In seven of these, there developed severe hypertension, the maximum systolic pressure ranging from 185 to 230 mm. (average, 210 mm.) of mercury and the maximum diastolic from 115 to 195 mm. (average, 140 mm.) of mercury; one of the eight had only moderate hypertension. This patient (V. K.), with a lesion at the fifth thoracic level, had a large capacity, hypotonic bladder with

considerable leakage around the catheter during experimental bladder filling. Six of the seven with severe hypertension experienced excruciating headache, of 9 + to 10 + intensity (in a 1 + to 10 + minimal-maximal scale), associated with peak levels of blood pressure; the other patient had only moderately severe (6 +) headache. The eighth, in whom only moderate hypertension was induced, also experienced headache of moderate intensity (5 +). In each patient headache intensity waxed and waned in close association with the rise and fall of blood pressure during the phases of bladder distention and deflation. In one of these eight subjects (E. K.) identical effects, including extreme rise in blood pressure and agonizing headache, developed when the bladder distention experiment was repeated on another occasion (three months later) in connection with a different manipulative procedure, aimed at modifying the headache so induced.

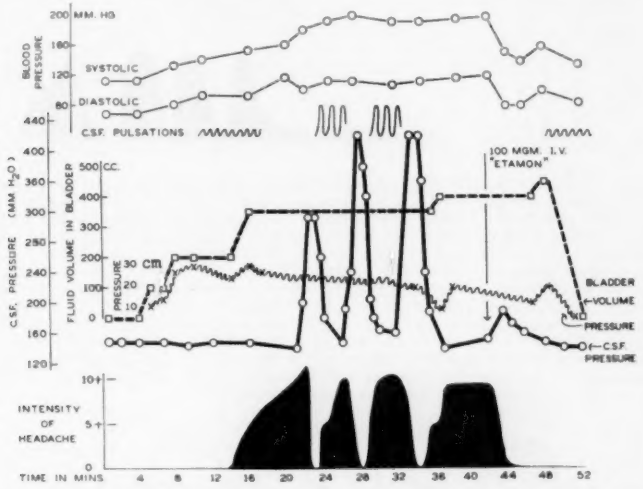

Chart 1.—Results of bladder distention in a subject (E. K.) with a complete transverse lesion of the cord at the eighth cervical level, showing effects both of artificially increasing the subarachnoid pressure and of injecting tetraethylammonium chloride intravenously in abolishing the headache induced by bladder distention. (In this experiment bladder pressure is expressed in centimeters of water, since a water manometer was used. In subsequent experiments, bladder pressures were continuously recorded cystometrographically in millimeters of mercury. Bladder pressure in this experiment was converted to millimeters of mercury in table 1.) Pulse rate, not shown here, decreased from 88 to 60 per minute in 32 minutes, rising abruptly to 108 per minute after the injection of tetraethylammonium chloride and falling slowly to 80 per minute thereafter. (Bilateral compression of the jugular veins prior to the beginning of bladder inflation, not illustrated, demonstrated rapid rise and fall between 160 and 440 mm. of water, indicating absence of spinal subarachnoid block.)

Of two patients with lesions at the eighth and tenth thoracic levels, respectively, a moderate rise of blood pressure in the first and no rise in the second, and no headache in either, occurred with bladder distention.

In four patients without spinal subarachnoid block, in whom severe headaches were induced by bladder distention, raising the intracranial pressure to 500 mm. of water by means of intrathecal injection of saline solution abolished headache completely, though all other factors remained constant, including volume and pressure

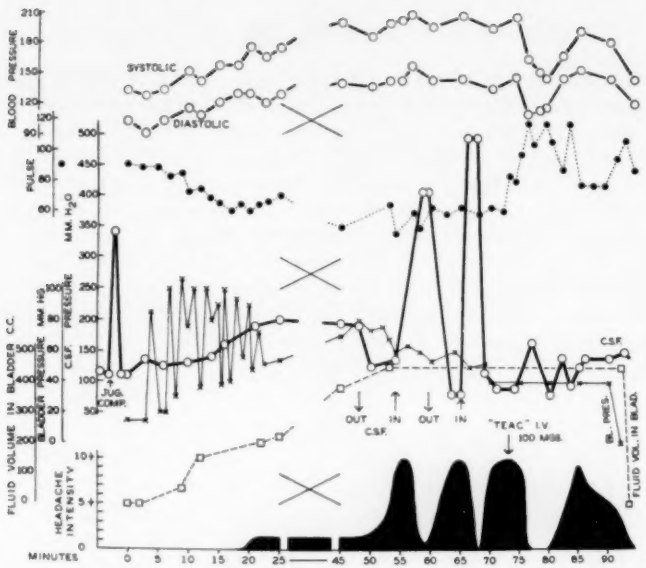

Chart 2.—Results of bladder distention in a subject with complete transverse lesion of the cord at the seventh thoracic level, showing, as in chart 1, steady rise in blood pressure and decline in pulse rate as the bladder fluid volume was increased. Because of failure of severe headache to develop after 45 minutes in association with a significant rise in blood pressure, a small amount of cerebrospinal fluid was drained from the spinal canal, with reduction in cerebrospinal fluid pressure from 200 to 120 mm. of water, followed shortly by rapid increase in intensity of headache which, as in other experiments, could be abolished at will by artificially increasing the subarachnoid pressure or injecting tetraethylammonium chloride intravenously. In this experiment the subsidence of the effect of the tetraethylammonium chloride was followed by the return of hypertension and headache during sustained bladder distention. After deflation of the bladder hypertension and headache promptly disappeared. The graph illustrates further the maintenance of induced hypertension and headache during sustained bladder distention, despite slow steady decline of increased intravesical pressure.

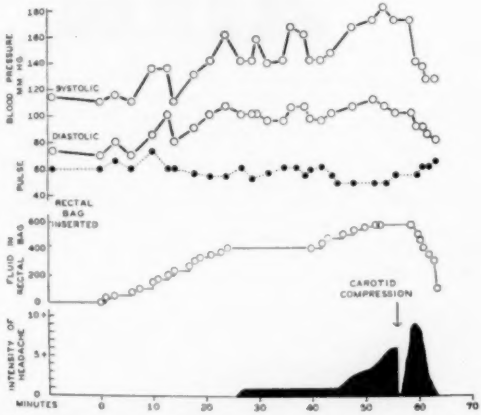

Chart 3.—Results of rectal distention in E. K. (same subject as in chart 1) with complete transverse lesion cord at the eighth cervical level. As in the case of bladder distention, maximal rise in blood pressure and peak intensity of headache were associated with maximal rectal distention, all effects disappearing with rectal deflation. The effect of bilateral digital compression of the carotid arteries in eliminating headache is shown, other factors remaining constant. Return of blood pressure to average levels and associated disappearance of headache following immediately on deflation of the rectal bag are shown.

of bladder fluid, elevated blood pressure, bradycardia and sweating (charts 1 and 2). Such headaches could be repeatedly reinduced in full intensity (during the course of sustained bladder distention and hypertension) by reduction of the cerebrospinal fluid pressure to normal. These patients had lesions at the fifth, sixth and eighth cervical and the seventh thoracic levels, respectively. In two additional patients, in whom complete spinal subarachnoid block was demonstrated by compression of the jugular veins, procedures identical with those leading to induced rise in intracranial pressure in the four preceding subjects led to no reduction in intensity of headache.

In two of three patients in whom bladder volume and pressure were maintained at high levels, headache was completely and instantly abolished by the intravenous injection of 100 mg. of tetraethylammonium chloride, with simultaneous reduction

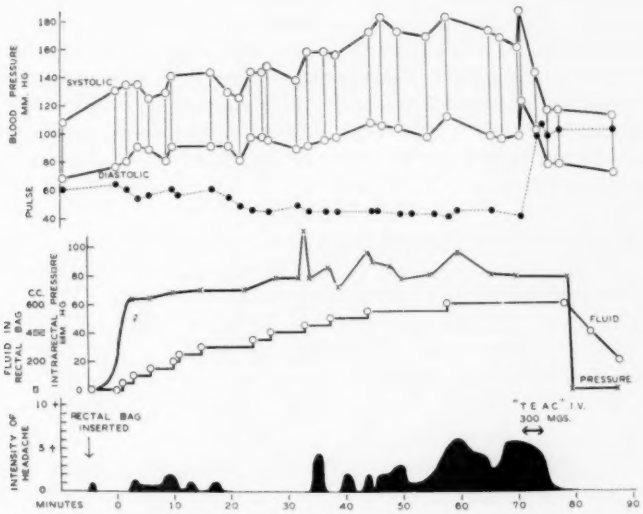

Chart 4.—Results of rectal distention in a subject with complete transverse lesion of the cord at the fifth cervical segment. In this subject (J. P., tables 1 and 2) bladder distention resulted in a greater magnitude of hypertension and greater intensity of headache than did rectal distention as illustrated here. Not indicated on the graph is the fact that at 55 minutes the application of digital pressure to the intrarectal bag resulted in simultaneous increase in headache intensity associated with further rise in blood pressure. The intravenous injection of tetraethylammonium chloride in three successive increments of 100 mg. each resulted in brief momentary rise in blood pressure after the first 100 mg., followed by rapid fall of blood pressure to average levels and coincident disappearance of headache.

of blood pressure to average, or slightly above average, levels (charts 1 and 2). In one of these patients headache and hypertension returned again to their former high level with the waning of the effect of the drug. In the third patient, headache was diminished in intensity, but not abolished, in association with a slighter reduction in blood pressure. In all three patients sweating and pilomotor reactions ceased. Bladder volume and pressure were not affected by the administration of the drug.

In two patients, bilateral compression of the carotid artery to the point of obliteration of palpable pulsations resulted in the complete elimination of headache in

each of five trials, its diminution in three trials and no effect in two trials. Systemic hypertension remained unaltered during compression of the carotid artery.

Of four patients, rectal distention in two (both with lesions of the cervical portion of the cord) led to effects identical with those resulting from bladder distention in the same patients. In one (chart 3), compression of the carotid artery completely eliminated headache transiently in each of two trials (only one trial shown), without alteration in systemic hypertension. In the other (chart 4), the intravenous injection of tetraethylammonium chloride (300 mg.) resulted in the same train of effects, including fall of blood pressure and elimination of headache, as that described with previous injections of this drug. During both procedures in which headache was eliminated, volume and pressure of the intrarectal fluid remained constant.

Two patients did not experience headache with rectal distention. In one, with a lesion of the cervical portion of the cord, moderate rise in blood pressure (to 160/106 mm. Hg), bradycardia, profuse sweating and pilomotor reaction developed. In a previous bladder distention experiment on this patient headache developed, but in association with a higher rise in blood pressure (215/195 mm. Hg, maximal). In the other patient, with a lesion at the tenth thoracic level, only slight rise in blood pressure (140/100), and no other autonomic effects, developed. In a previous experiment on this patient, bladder distention similarly failed to produce significant hypertension or headache.

COMMENT

In these experiments, when the lesion of the cord was at a high enough level, the introduction of fluid into the bladder at a rate more rapid than that at which urine normally accumulates resulted very soon in changes in pulse and blood pressure, sweating and, later, headache, all of which became maximal at the point of maximal filling, but not invariably at the point of maximal pressure. Save for the fact that distention of the bladder and increase in the intravesical pressure led to these effects, there was no intimate correlation between either bladder volume or bladder pressure, on the one hand, and the resulting effects, on the other.

Bladder volume was routinely increased, either steadily or in stages, to the point of presumed maximal capacity, namely, to the point when fluid no longer continued to flow from the reservoir. The latter was elevated 80 to 100 cm. above the bladder, and fluid was allowed to flow from the reservoir by a Murphy drip at rates which varied during successive stages in the experiment, depending on the effect being obtained or the spontaneous rate of acceptance of fluid by the bladder with the clamp entirely open. Thus, early in each experiment, fluid was usually allowed to enter rapidly (generally from 30 to 50 cc. per minute). This resulted in some instances (not correlated with the level of the lesion or the ultimate capacity of the bladder) in the immediate appearance of strong bladder contractions, which of themselves temporarily arrested further inflow; in other instances, in a slow steady rise in bladder pressure with proportionately slower rate of acceptance of fluid. In all cases, finally, no further fluid entered the bladder. This was taken as the bladder capacity. Filling was frequently interrupted for periods of minutes while the blood pressure was rising, until the effects produced became static or diminished, when further filling was induced. During such periods pressure as recorded on the cystometrogram sometimes remained steady, even though reflex effects were declining.

Analysis of the ink-recorded cystometrographic records obtained on different subjects showed that wide differences in levels of bladder pressure were capable of producing maximal reflex effects. E. K. never had an intravesical pressure over 20 mm. Hg of mercury, even during maximal filling and maximal headache. J. W., on the other hand, with early powerful bladder contractions and pressures of 70 to 80 mm. Hg, associated with rapid rise in blood pressure and other effects, maintained these changes in intervals between contractions when the bladder pressure was as low as 20 mm. of mercury. Repeatedly it was noted that increasing the fluid volume led to further increase in reflex effects, though the ultimate intravesical pressure was no greater, and sometimes slightly less (chart 2). This suggests that effective afferent stimuli from the bladder wall depend essentially on the degree of stretching of the bladder wall and the distortion of its end organs, which is a function not of intravesical pressure alone but also of the degree of relaxation of the bladder wall.

The pathways, peripheral and central, by which afferent impulses from the bladder ultimately reach effector organs may be analyzed on the basis of available evidence. It has been assumed, since the work of Head and Riddoch, that the area of integration for such reflex effects lies within the segments of the distal stump of the spinal cord. The question may be raised whether afferent impulses from the bladder, via either the sacral roots or the hypogastric plexus, do not pass to the cells of the paravertebral sympathetic ganglia directly to excite postganglionic sympathetic neurons, with resultant sudomotor, pilomotor and vasoconstrictor effects. This would seem highly unlikely in view of the participation of skeletal muscle contraction in the "mass reflex" of Head and Riddoch. In the experiments here reported powerful muscle spasms of the lower extremities and trunk occurred repeatedly in association with bladder distention, thus indicating reflex excitation of anterior horn cells in cord segments. In addition, as Head and Riddoch have pointed out, stimulation of other receptor surfaces, such as the skin of the lower extremities, leads to a similar mass discharge. Such afferent impulses undoubtedly enter the spinal cord via somatic nerves and posterior roots to produce reflex effects. Lastly, direct synaptic connections between visceral afferent fibers and postganglionic cells in paravertebral sympathetic ganglia have not been proved to exist (when experiments were carried out with unquestionably decentralized ganglia).

Further clinical evidence supporting the concept that the intact segments of the distal stump of the spinal cord are the areas of central integration for the reflex effects initiated by bladder or rectal distention is the failure of occurrence of these effects with rectal distention in a subject who had had ascending myelitis, becoming finally stationary at the first thoracic level. It was evident that the distal, involved portion of the cord in this patient was destroyed during the course of the ascending inflammatory process.[6]

The peripheral route of afferent impulses from the stretched bladder wall was considered by Guttmann and Whitteridge to be in the small unmyelinated fibers of the hypogastric nerves, since it had been previously found that impulses in these fibers continue as long as steady pressure is maintained in the bladder. Since the

6. Robertson, H. S., and Wolff, H. G.: Studies on Headache: Distention of the Rectum, Sigmoid Colon and Bladder as a Source of Headache in Intact Human Subjects, Arch. Neurol. & Psychiat. **63**:52 (Jan.) 1950.

bladder has a double afferent innervation, an alternate possible pathway is the pelvic nerve entering the cord via the posterior roots of the second, third and fourth sacral segments. Thompson and Witham [3] found that relatively small amounts of procaine injected into the lumbar subarachnoid space prevented the reflex effects of bladder distention, whereas bilateral lumbar sympathetic block did not. These data support the concept that afferent impulses are conveyed by sacral roots to the sacral portion of the spinal cord with cephalad spread of the excitation process within the intact distal stump to autonomic effector neurons (preganglionic) in the lateral horn of the thoracolumbar segments, resulting in a mass outflow from these segments. As has already been suggested by previous authors, such mass discharges are undoubtedly made possible by the loss of inhibiting influences from higher centers.

As may be seen from table 1, all but one of the patients whose cord transections were at the seventh thoracic level or above exhibited marked rises in blood pressure in response to bladder distention. Subject J. L. had the lowest lesion (seventh thoracic level) of those who exhibited significant responses. Though the site of the lesion was not verified by laminectomy, a sensory level at the seventh thoracic dermatome, below which all forms of sensation were lost, and x-ray evidence of bony destruction at the seventh and eighth thoracic vetebrae from a previous bullet wound indicated that the presumed site of transection of the cord was certainly no higher than the seventh thoracic segment. The marked effect on blood pressure is in contrast to that of Guttmann and Whitteridge's patients, with lesions below the fifth thoracic segment. None of these had significant rises in blood pressure. It is of interest that, though J. L. exhibited profuse sweating, the area of sweating was sharply delimited to below the twelfth thoracic dermatome, involving only the lower extremities. Among Thompson and Witham's six patients, two with lesions below the fifth thoracic segment (seventh thoracic and second lumbar, respectively) did not have rises in blood presure; but, as in our subject, the patient with the lesion at the seventh thoracic level had sweating in both lower extremities.

Though final conclusions cannot be drawn in the absence of verification of the site of the lesion by direct inspection in the subject mentioned, it would appear that sympathetic discharges capable of producing significant vasoconstriction and hypertension may emanate from segments lower (possibly the sixth and seventh thoracic) than the heretofore accepted lower limits of the fifth thoracic. Head and Riddoch's patient, with a lesion at the seventh thoracic segment, who had sweating of the face and neck from bladder distention, affords further evidence for the possibility of sympathetic effects in areas derived from levels of the cord lower than those commonly thought to provide the innervation for these effects. However, it was Guttmann and Whitteridge's opinion that the sweating in this case could not "be attributed to activity of the fibers arising from the part of the cord below the level of the lesion," but was due to a "thermoregulatory response mediated by efferent fibers arising from above the spinal cord lesion."

Though the lower limit of lesions of the neuraxis permitting reflex vasoconstriction and hypertension as a result of noxious impulses from the viscera seems roughly established, the upper limit of lesions permitting such abnormal reflexes has not been defined. It has been assumed, since such reflexes have not previously been demonstrated when the neuraxis was intact, that inhibitory impulses from suprasegmental areas prevent them. Whether such centers lie in the intracranial neuraxis, i. e., the

brain stem, or in the region of the diencephalon (thalamus, hypothalamus) and basal ganglia, or at still higher (cortical) levels, is not known. In the papers referred to above, patients were not studied whose lesions were above the cervical level of the spinal cord. Though in spinal cats, just as in spinal man, the blood pressure rises with distention of the alimentary canal, it has been reported that in decerebrate cats the blood pressure falls.[7]

An opportunity to study the results of bladder distention in decerebrate man presented itself in J. M., a white man aged 24. After a severe craniocerebral injury seven months prior to the experiment, the subject had been under observation in the hospital, presenting a picture of total amentia and incontinence of urine and feces and requiring daily tube feedings. He appeared totally unaware of, and unresponsive to, his surroundings. All four limbs exhibited fluctuating extensor rigidity with periodic brief tonic seizures. Fragmentary tonic neck (Magnus-de Klijn) reflexes could be demonstrated. Pronounced pupillary and oculomotor abnormalities indicated a severe lesion of the midbrain. Pneumoencephalography confirmed the presence of cerebral atrophy.

Bladder filling to a capacity of 470 cc. and a pressure of 85 mm. Hg resulted in a rise in blood pressure from 150/86 to 192/120 mm. Hg, an increase in pulse rate from 100 to 130 per minute and profuse sweating over the head, neck and arms. Emptying the bladder led to disappearance of all effects. Thus, abnormal spinal reflexes resulting from bladder distention were not inhibited when the lesion was as high as the midbrain, indicating that the inhibitory centers preventing such effects in the intact subject lie above the level of the midbrain.

The close correlation between induced hypertension and the development of headache in patients with lesions of the cord, along with the occurrence of superficial vasodilatation in the head (skin and nasal mucosa), suggests that the mechanism of such headache is the distention of pain-sensitive cranial arteries. Such distention would appear to be due to the heightened intravascular tension caused by reflex vasoconstriction elsewhere (limbs and visceral bed). The transitory complete or partial elimination of the headache attendant on compression of the internal carotid arteries in the majority of trials supports this concept. The complete elimination of the headache by artificially raising the intracranial pressure in all trials in all subjects when the presence of subarachnoid block did not preclude this procedure further substantiates the proposed mechanism of production of head pain and suggests that intracranial (pial) arteries are the chief site of painful dilatation. The immediate fall in blood pressure and concomitant cessation of headache resulting from the injection of tetraethylammonium chloride would indicate that paralysis of the vasoconstrictor reflex with drop in blood pressure permits reduction in the degree of distention of the cranial arteries to below pain threshold levels.

SUMMARY AND CONCLUSIONS

The occurrence of abnormal autonomic reflexes as a result of bladder or rectal distention in subjects with transverse injuries of the spinal cord is confirmed.

Effective afferent stimuli from the bladder wall depend essentially on the degree of stretching of the wall and on the distortion of afferent end organs. These changes

7. Irving, J. T.; McSwiney, B. A., and Suffolk, S. F.: Afferent Fibres from Stomach and Small Intestine, J. Physiol. **89**:407, 1937.

are a function not of intravesical pressure alone but of the degree of relaxation of the bladder wall as well.

The areas of central integration for the reflex effects initiated by bladder or rectal distention are the intact segments of the distal stump of the spinal cord.

The peripheral route of afferent impulses from the stretched bladder wall to the spinal cord is probably via the posterior roots of the sacral segments.

Hypertension developing in response to bladder distention in subjects with lesions at or above the fifth thoracic level is confirmed. Further, hypertension was shown to be similarly induced in a subject with a transverse lesion of the cord as low as the seventh thoracic level.

Suprasegmental inhibitory centers preventing such reflex effects lie above the level of the midbrain.

Headache experimentally induced in subjects with transverse lesions of the cord by bladder or rectal distention may be completely abolished by artificially increasing intracranial pressure, by bilateral compression of the carotid artery and by lowering the reflexly induced hypertension, either through injection of tetraethylammonium chloride intravenously or through terminating bladder or rectal distention.

Reflex discharges from the distal stump of the spinal cord originating from afferent end organs of the stretched bladder and rectal wall result in vasoconstriction of the limbs and visceral bed sufficient to elevate the systemic arterial pressure, with secondary passive distention of cranial arteries causing headache.

EXPERIMENTAL PHYSIOLOGICAL STUDIES WITH LYSERGIC ACID DIETHYLAMIDE (LSD-25)

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STOLL [1] studied the effects of lysergic acid diethylamide (LSD-25) in both psychotic and normal subjects. Condrau [2] and others reported their results of administration of lysergic acid diethylamide to various types of subjects and in general confirmed the findings of Stoll.

For details of the chemistry of the ergot alkaloids, we refer to a more recent, comprehensive work by Stoll.[3] It should be mentioned in passing that all lysergic acid alkaloids have lysergic acid as a base. This substance is a multiple, active, polycyclic, nitrogenous carboxylic acid, which so far has not been synthesized. The natural ergot alkaloids contain *d*-lysergic acid and may be divided into two groups—the ergotamine-ergotoxine group, on the one hand, and the ergonovine group, on the other. In the former group *d*-lysergic acid is combined with a manifold peptid, whereas the latter group comprises the mono acid amides of the *d*-lysergic acid. Of this group only one natural representative is known, ergonovine, or *d*-lysergic acid-1-isopropanolamide. The substance under study (LSD) is *d*-lysergic acid diethylamide, the synthetic amide of the organic *d*-lysergic acid with a secondary amine, diethylamine. It, therefore, belongs to the ergobasine group, of which it is a partial synthetic representative. It was obtained for the first time in 1938 (W. Stoll and A. Hofmann).

Lysergic acid produces profound psychic effects in extremely small quantities. Ten to 60 micrograms of the drug is usually enough to produce marked psychic changes, with euphoria, depression and, as reported by Stoll, pupillary changes, alterations in tendon reflexes and visual hallucinations. Although various physiological changes have been attributed to the administration of this substance, no controlled studies on this phase could be found. In an effort to clarify the physiological effects of lysergic acid diethylamide, the following studies were carried out.

PRESENT INVESTIGATION

Material and Methods.—Six male schizophrenic patients were selected on the basis of poor prognosis and failure to respond to other types of therapy. One had been hospitalized for one

From the Ypsilanti State Hospital.

Lysergic acid diethylamide was supplied through the courtesy of Sandoz Chemical Works, Inc., New York.

1. Stoll, W.: Schweiz. Arch. f. Neurol. u. Psychiat. **60**:279, 1947.
2. Condrau, G.: Acta psychiat. et neurol. **24**:9, 1949.
3. Stoll, W.: Schweiz. med. Wchnschr. **79**:110, 1949.

year; three, for five years; one, for two years, and one, for 34 years. For four of the six patients the diagnosis was paranoid schizophrenia, and for the other two, hebephrenic schizophrenia. A complete physical examination, electroencephalographic and electrocardiographic recordings, blood sugar, nonprotein nitrogen determinations, white blood cell count, hemoglobin determination, urinalysis and cephalin-cholesterol flocculation tests were carried out before the initiation of the experiment and the values found to be within normal limits.

Conditions of Study.—Patients were studied in a room where a trained nurse and physician were in constant attendance. A control period, during which the blood pressure, pulse and respiration rates, deep reflexes, pupillary size and degree of salivation were determined every half-hour, showed no significant alterations in physiological status. Additional control studies were carried out with other substances. Epinephrine hydrochloride in oil, 1 cc., was administered intramuscularly to each of these six patients. The blood pressure and pulse and respiration rates were averaged, and it was determined that the patients acted in the usual manner to this drug.

Lysergic acid diethylamide was administered by mouth in ascending doses, beginning with 0.5 microgram and reaching a maximal of 6 micrograms per kilogram of body weight.

Results.—When the blood pressure responses of these six patients were averaged for each dosage level, the following results emerged: When lysergic acid diethylamide was administered orally in doses up to 6 micrograms per kilogram, it produced a slight rise of blood pressure in all patients. Blood pressure curves were entirely similar during the maximal height of response 2½ hours after administration and returned to normal five or six hours after administration. Although a slight increase in systolic blood pressure was noted, all changes in blood pressure were within normal physiological limits. Except at a dosage level of 2 micrograms per kilogram of body weight, an increase in pulse rate was noted with the administration of increasing amounts of the drug. The maximal increase in pulse rate occurred between one and two hours after administration and gradually declined to pretreatment levels five to six hours after administration. The average pulse rates were always within normal physiological limits, but there was wide variation in individual subjects. One subject showed a depression, and another a decided increase, in the pulse rate after administration of the drug. The extremes, in general, were in the order of 100 per minute as a maximum and 48 as a minimum. Lysergic acid diethylamide in doses up to 6 micrograms per kilogram by mouth does not appear to be a respiratory depressant. There is, on the contrary, a suggestion that it may be a respiratory stimulant, but our present experimental data are too limited to permit formulation of any definite conclusion. For example, with 6 micrograms of lysergic acid diethylamide per kilogram of body weight, one patient showed a respiratory rate of 32 per minute (the highest recorded) and another a respiratory rate of 12 per minute (the lowest recorded).

Administration of lysergic acid diethylamide by mouth in ascending doses up to 6 micrograms per kilogram produced constant flushing responses, which had, however, considerable variation in intensity and duration in different subjects. Duration, as well as rapidity of onset and degree of flushing, seemed to be related to the size of the dose. When the drug was administered by mouth, increased salivation was produced. In no case was dryness of the oral mucous membranes noted. We found, in studying these six patients, that the higher the dose of lysergic acid diethylamide, the greater the salivation response. Individual responses to administration of the drug showed considerable variation in degree but always followed a pattern of quantitative increase in salivation and longer duration of response with increase in dose.

Contrary to the findings of other investigators, nausea, vomiting and anorexia were not prominent in the present group of patients. In a total of 42 treatments, vomiting was experienced three times. Lysergic acid diethylamide, when administered by mouth in the doses noted, regularly produced dilation of the pupils. As the dose was increased, the reaction to light was progressively impaired in some persons. Maximal pupillary dilation was obtained from two to three hours after administration, the pupillary size gradually returning to normal. The degree of pupillary dilation and duration of this response were directly related to the size of the dose. Conjunctival injection was noted to occur to a varying extent with the lower dosage levels, but with 6 micrograms per kilogram all six patients showed conjunctival injection, which varied only in degree. Increased lacrimation paralleled the increase in dose. With this small series of patients no definite conclusions can be drawn concerning the febrile effects of lysergic acid diethylamide. Two of five patients showed a rise in temperature of 1 degree (F.) when 2 micrograms per kilogram was administered by mouth. Lysergic acid diethylamide produced in these six patients a constant hyperreflexia. The degree of hyperactivity of the knee jerks was directly related to the size of the dose. With 6 micrograms per kilogram, hyperactivity of the deep reflexes was constant in all patients and was present in a far greater degree than when smaller doses were used. The Babinski sign was not observed. There was slight unsteadiness of gait. There was no disturbance of coordination, no past pointing phenomena and no nystagmus. These findings are at variance with those of others, who have noted hypoactive reflexes, nystagmus, ataxia with a positive Romberg sign and past pointing phenomena.

Blood sugar curves without medication showed no significant variation during the fasting period. The responses of these six patients to the administration of 0.5 cc. of epinephrine hydrochloride (1:1,000) was measured by obtaining the blood sugars. The rise in blood sugar was noted 15 minutes after administration of the epinephrine. The level returned to normal, only to rise again to even greater height one hour after administration. These findings are in keeping with the previously determined blood sugar responses to epinephrine in normal subjects. In the present group of patients there was a slight rise in blood sugar beginning with the second or third hour after administration of lysergic acid diethylamide by mouth in a dose of 1 microgram per kilogram of body weight. Average blood sugar values ranged from 91.3 mg. per 100 cc., before administration of the drug, to 97.3 mg. per 100 cc., four hours after administration. With the dose of 1 microgram of the drug per kilogram given by mouth, there was a slight rise in fasting blood sugar, insufficient to permit any conclusion.

Lysergic acid diethylamine when administered in doses of 1 microgram per kilogram produced definite leukocytosis. Provisional conclusions based on this experiment indicate that there is an evaluation of the white blood cell count as a result of administration of the drug (table). The reasons for these alterations are not known.

Lysergic acid when administered in the dose of 1 microgram per kilogram every three to four days for a total of six doses produced no urinary changes. The pretreatment cephalin-cholesterol flocculation tests on all patients gave negative results. During treatment with the lysergic acid these tests continued to give

negative results. We conclude, therefore, that there is no change in hepatic function as a result of administration of the drug in this amount.

Nonprotein nitrogen values before treatment were within normal limits. Nonprotein nitrogen values during the course of treatment were within normal limits and showed no alteration in individual cases. Immediately after the termination of this experiment nonprotein nitrogen estimates showed no change from pretreatment levels. Six weeks after administration of the last dose of lysergic acid diethylamide nonprotein nitrogen values were likewise normal.

The white blood cell and hemoglobin content were determined before treatment, during treatment, immediately after treatment and six weeks after administration of the last dose of the drug. The pretreatment white blood cell count and hemoglobin content were normal for all patients and remained so during the course of treatment, immediately after treatment and six weeks after treatment. It should be noted that these determinations were made on nontreatment days.

White Blood Cell and Differential Counts with Administration of 1 Microgram of Lysergic Acid Diethylamide per Kilogram

Patient	White Blood Cell Count	Basophils	Eosinophils	Myelocytes	Juvenile Forms	Stab Forms	Segmented Forms	Lymphocytes	Monocytes	Degenerated Forms
Before administration										
1	5,800	..	1	..	..	2	74	21	2	..
2	4,950	(differential not done)			..	..	..	..	..	..
3	7,550	1	4	..	..	2	65	22	6	3
4	6,600	..	3	..	..	..	41	53	3	..
5	9,300	1	1	..	2	..	64	29	3	..
Three hours after administration										
1	7,300	2	2	..	..	..	59	36	1	1
2	6,250	1	8	..	..	..	53	38	..	..
3	8,800	..	1	..	..	..	79	19	..	..
4	7,150	1	1	..	..	..	42	56	..	..
5	22,450	..	2	..	..	..	76	22	..	..
Six hours after administration										
1	5,100	1	3	2	..	..	61	33	..	..
2	6,950	..	6	..	..	1	43	50	..	..
3	9,100	2	1	..	..	..	54	39	4	..
4	11,050	2	2	..	1	..	57	36	2	..
5	15,100	..	2	..	..	..	55	38	5	..

Lysergic acid diethylamide as administered had no effect on the weight of five of six patients. One patient showed a gain in weight of 20 pounds (9.1 Kg). Direct instillation of lysergic acid diethylamide into the conjunctival sac (approximately 1 microgram per eye) produced very little effect on the pupils. There was, however, evidence of dilation of slight degree. It becomes evident from this result that pupillary dilation observed during the administration of lysergic acid diethylamide may be due primarily to the central effects of the drug.

Electroencephalographic responses to the administration of 2 micrograms per kilogram of lysergic acid diethylamide were determined. No changes from premedication electroencephalograms could be discerned. In these six patients, who were administered ascending doses of lysergic acid diethylamide, a psychic response, particularly in regard to accessibility, hallucinations, delusions, affectivity and general demeanor, was observed.

When 0.5 microgram of lysergic acid diethylamide per kilogram was administered, definite psychic changes were noted in five of six patients. All changes began one-half to one hour after administration and reached their height two to 2½ hours after administration. These effects gradually subsided, and the patients had returned to their customary psychic status four to five hours after administration

of the drug. The first changes noted were euphoria, increased spontaneous verbal productivity and alteration from a surly, antagonistic attitude toward the physician to amiability, talkativeness, euphoria and outbursts of laughter. At the height of the reaction patients were laughing in what was felt to be an appropriate manner and were definitely more accessible. One patient showed slight depression between euphoric outbursts. Another became hyperactive, very disturbed and hostile toward the physician, complained of dying and made multiple requests to go home. After the observation period he was noted to be drinking large quantities of water and to be inducing vomiting. A three minute "seizure" occurred in the evening of the same day. During the following night he was incontinent of urine, after which he had an increase in temperature to 100.4 F. and complained of headache. No hallucinations were elicited in this group of patients with the dose indicated.

When 1 microgram of lysergic acid diethylamide per kilogram was administered by mouth, the above observations were confirmed, the effects being greater in degree and more prolonged in duration. Euphoria was prominent. No instance of depression was noted. One patient complained of being worked on by "magic" and expressed the fear that his penis would dry up. He became agitated and complained of "seeing magic." Three of six patients experienced hallucinations with this size of dose. Increased alertness and amiability were observed. When the experiment was repeated with the same size of dose, the same general response was noted. Hallucinations were noted in two of five patients so treated. These were of the "primary" type previously described by others as occurring under the influence of lysergic acid diethylamide.

When 2 micrograms of lysergic acid diethylamide per kilogram was administered by mouth, the same general reaction patterns were observed. Patients were more alert and showed outbursts of euphoria. One patient complained of various unusual somatic sensations. The patients in general were more amiable and more easily accessible. Misidentification of other persons in the environment was noted. Two of five patients admitted to having hallucinations. In one an increased volume of delusional material was noted. Outbursts of infectious laughter were prominent.

When 4 micrograms of lysergic acid diethylamide per kilogram was administered by mouth, the same general reactions were observed. However, these reactions were more prolonged and more intense with the increased dose. Hallucinations were noted in two of six patients.

When 6 micrograms of the drug per kilogram was administered by mouth, the same general reactions were noted. Hallucinations were noted in two of five patients. Psychomotor retardation was in evidence. The patients always maintained relatively good contact with their environment. It should be noted that hallucinations occurred in all patients at some time during these administrations of lysergic acid diethylamide. However, hallucinatory experiences often could not be determined while the patient was under the influence of the drug, but could be elicited some time afterward. In order of frequency, there were first visual hallucinations, consisting of patterns, diagrams and flashes of light, and then paresthesias, with sensations of elongation of the limbs, crawling over the skin and various other somatic sensory disturbances. One patient experienced olfactory hallucinations. Euphoria was prominent in all patients, occurring usually as outbursts, between which patients appeared somewhat disinterested and apathetic.

Only one patient showed evidence of depression, and then for a short time. Increased accessibility, increased spontaneity, clearer verbal productivity and increase in delusional formulations, increased psychomotor activity and elements of agitation were also noted. Ataxia was not prominent and was seen in only one patient, as evidenced by difficulty in walking and eating with a fork and spoon. No past pointing phenomenon or other disturbances of coordination were observed. The patients were observed particularly in regard to their production of sexually determined material. With the increase of spontaneity noted in these patients as a result of administration of the drug, increased spontaneous sexual material was quite apparent in three patients. One patient, for example, recalled a homosexual episode which he had as a youth and which had not previously been accessible. Another talked about his delusion of having been raped by a monkey, and a third was constantly asking the nurse who was in attendance, "You're my sister; marry me?" Another patient would ask whether the nurse would engage in sexual intercourse. We believe that the increase in accessibility and spontaneity as a result of administration of lysergic acid diethylamide is a general reaction phenomenon and that there are no specific isolated facets of the personality in which retardation or blocking occurs. Although the group of patients is small and the number of treatments limited, it becomes apparent that this increased spontaneity and accessibility is a general psychic reaction; and since sexuality is a part of personality structure, it is only to be expected that sexual material will become more evident with increased spontaneity and productivity.

Two patients who were totally blind, one from methyl alcohol poisoning and the other as a result of a gunshot wound severing the optic nerves, were administered lysergic acid diethylamide to the amount of 1 microgram per kilogram. Their reactions were noted at frequent intervals. Neither patient was manifestly psychotic at the time of administration of the drug. Euphoria and hypersalivation were noted in these patients. One became paranoid, an element which was only in slight evidence before administration of the drug. He denied that he had received any special drug and thought that he had been given only water. He possibly had olfactory hallucinations, or at least misinterpreted stimuli, since he believed that the tobacco smoke which he smelled in the air was really opium. He denied having any visual hallucinations. The second patient became euphoric and showed increased hyperactivity of the deep reflexes and slight dilation of the right pupil. He noted increased lacrimation and slightly increased moisture of the mucous membranes of the mouth. He showed more spontaneous verbalization and denied having any hallucinations. Although this experiment was carried out with only two patients, it was noted that both gave the same general reaction to the drug. There was an increased accessibility, increased euphoria, increased salivation and lacrimation and increased tendon reflexes, but no visual hallucinations, with this dose. It should be noted that both patients had previously had their eye sight and were not congenitally blind.

Because of the increased secretory response noted as a result of administration of lysergic acid diethylamide, the response in the atropinized human subject was determined. These patients were given 1 mg. of atropine sulfate by intramuscular injection 4½ hours prior to the oral administration of 6 micrograms of lysergic acid diethylamide per kilogram. In general, there was slight flushing of the skin in all patients, beginning one to two hours after oral administration of 6 micrograms of

lysergic acid diethylamide per kilogram. The flushing response was somewhat irregular, but the skin color of most patients had returned to normal six hours after administration. In general, the mucous membranes of the mouth were moist. No drying was noted in any of the patients. One patient showed slightly increased salivation beginning 2½ hours after administration of the drug, and others maintained moist mucous membranes more or less constantly for four to five hours after its administration. The pronounced drying response of this dose of atropine was not maintained when lysergic acid diethylamide was administered subsequently. All patients showed an initial dilation of the pupils, which was increased by the further administration of lysergic acid diethylamide. In general, the maximal pupillary dilation was noted between one and 2½ hours after administration of the drug. Pupillary signs gradually decreased to normal until six hours after administration of lysergic acid diethylamide, after which no effect of either medication was noted.

In general, there was increase in the deep reflexes beginning 1½ hours after administration of the drug, reaching a maximum at approximately two to 2½ hours and gradually declining to normal in six hours. This response was somewhat irregular and varied from patient to patient, but in general there was an increase in deep reflexes, in some patients being very pronounced. The Babinski sign was not elicited. All patients were cooperative during the entire proceeding. All were able to eat the noon meal during the course of the experiment. All patients showed much more spontaneity; euphoric outbursts were prominent. Increased tearing was noted in several. Euphoria seemed to be more prolonged and more sustained than with the same dose of lysergic acid diethylamide without atropine. Hallucinations occurred in most patients, only one failing to have this reaction. Increased accessibility and amiability were noted in all patients. Increased urinary frequency was not observed except in one patient, who during the first hour after administration of lysergic acid diethylamide micturated three times. Slight ataxic phenomena were in evidence but were never so severe as to interfere with the patient's performing such acts as buttoning the clothing, eating, standing, walking and drinking. We concluded from this single experiment with a small group of patients that premedication with atropine decreased the salivation response noted when lysergic acid diethylamide was administered alone. Also to be noted was more persistent euphoria than had been seen previously with this dosage level. Because of the small number of patients, the latter effect cannot be adequately evaluated.

All these six patients were in such poor contact with their environment that very few responses could be elicited in regard to their subjective feelings while under the influence of the drug. One patient felt that his mouth had "cotton in it"; another, that he had been given "magic," and still another complained of a "constriction in my chest." It has been noted that the barbiturates administered to patients under the influence of lysergic acid diethylamide abolish the psychic effects of the latter drug. It is well known that barbiturates initially act on subcortical structures. The site of action of lysergic acid diethylamide is not known. We consider, however, that the drug acts primarily on the cortex to produce a depression, and there is abundant evidence to suggest this, i. e., increased deep reflexes, dilation of the pupils, salivation, euphoria and increased accessibility. One might think of the effects of lysergic acid as being due to a release of the lower centers from cortical control. It would hold, then, that any drug which depresses the subcortical centers would, by blocking the subcortical release effectively, nullify the psychic action of

lysergic acid diethylamide. This would be further evidence for and support of our present belief that lysergic acid diethylamide in the amounts used acts primarily on the cortex, that the neurological symptoms following administration are directly attributable to this cortical effect and that the psychic phenomena witnessed under its influence are the result of subcortical discharges no longer under the full control of the cortex.

From studies carried out on animals, lysergic acid has been determined to be a relatively nontoxic substance. The lethal intravenous dose was 65 mg. per kilogram and the lethal subcutaneous dose 285 mg. per kilogram in laboratory animals. Our maximum dose was 6 micrograms per kilogram. It would seem therefore that lysergic acid diethylamide is an extremely safe and relatively nontoxic drug.

CONCLUSION

Studies with lysergic acid diethylamide (LSD-25) have been carried out in an effort to clarify the physiological and psychic responses attendant on administration of this drug in schizophrenic patients. The drug produced slight increase in blood pressure, slight increase in pulse rate, no essential change in respiration, increase in salivation and lacrimation, dilation of the pupils, increase in deep reflexes and slight ataxia. Oral administration produced pupillary dilation of marked degree, whereas topical administration produced very slight dilation. The total white blood cell count was increased during the time of action of the drug. Euphoria occurring in outbursts, was prominent. Increased accessibility and amiability, with increased release of libido and greater accessibility of delusional material, was observed. Visual hallucinations of the so-called primary type were not noted in two blind patients treated with the drug but were seen in all of the six patients on whom complete studies were carried out. Urinary constituents, the nonprotein nitrogen level, the electroencephalogram, cephalin-cholesterol flocculation, weight and temperature were not affected by the administration of this drug in doses up to 6 micrograms per kilogram. Lysergic acid diethylamide appears to be a suitable substance for further therapeutic investigation in the psychoses.

SIGNIFICANCE OF RISE IN BLOOD SUGAR LEVEL AFTER INJECTION OF EPINEPHRINE IN MENTAL DISEASE

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MEASUREMENT of the rise in the blood sugar level after the injections of epinephrine has been used for many years as a means of estimating glycogen stores in the body [1]; it is believed that all of the glycogen which is broken down to glucose during the test is derived from the liver, for epinephrine does not influence the glycogen content of muscle in man.[2] Evidence is available indicating that an excess of adrenocortical hormones, which favor glycogen formation, is present in patients with mental disease,[3] and therefore it was considered desirable to study the rise in blood sugar after injection of epinephrine in treated and untreated patients with mental disease in order to extend the earlier observations of others [4] in this respect.

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(Footnote continued on next page)

MATERIAL AND METHODS

Fifty-one untreated patients, ranging in age from 16 to 50 years, were studied shortly after admission; 33 were women. The diagnosis of a severe neurosis was made for 10; 35 were considered to be suffering from schizophrenia, and 6 had manic-depressive psychoses (chart 1). With four exceptions, all of the patients studied were grossly underweight or had lost from 5 to 30 per cent of their weight or both.

Epinephrine hydrochloride was injected in doses of 0.01 mg. per kilogram of weight; blood was drawn before and, again, 30, 45 and 60 minutes after the injection and analyzed for the sugar content by means of the method of Folin and Wu.[5] Twenty-one additional tests were done after the end of treatment with insulin (patient ambulatory) or with electric shock.

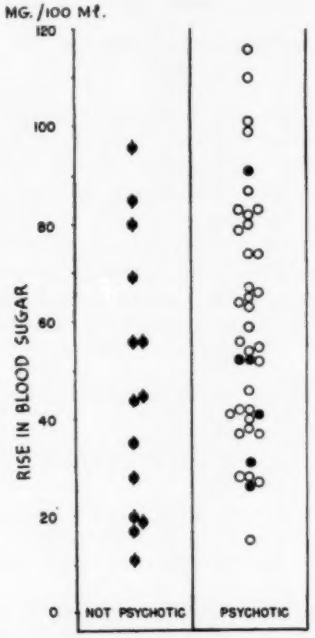

Chart 1.—Rise in blood sugar after injection of epinephrine hydrochloride (0.01 mg. per kilogram) in anoretic, malnourished patients with mental disorders.

Values for patients with neuroses are indicated by black circles with a vertical line; values for patients with schizophrenia, by clear circles, and values for patients with other psychoses, by black circles.

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5. Folin, O., and Wu, C., in Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry: Volume II, Methods, Baltimore, Williams & Wilkins Company, 1932, p. 456.

OBSERVATIONS

The maximal rise in blood sugar after the injection of epinephrine in untreated patients ranged from 11 to 116 mg. per 100 ml. of blood. The range in patients with neuroses was between 11 and 85 mg., with two thirds of the values over 20 mg. per 100 ml.; for the psychoses studied the range was between 15 and 116 mg., with 98 per cent of the values above 20 mg. per 100 ml. The measurements for psychotic patients were sufficiently numerous to plot the distribution, and a typical bell-shaped curve was obtained, with the peak consisting of 13 readings in the range between 40 and 59 mg. per 100 ml. There appeared to be no differences related to the various diagnoses. There is no evidence in this respect that schizophrenia causes a response to epinephrine that is less than that which occurs in other types of mental disease or in normal subjects.

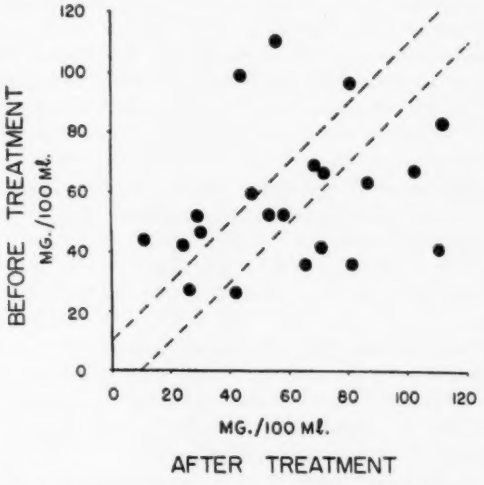

Chart 2.—Rise in blood sugar after injection of epinephrine hydrochloride (0.01 mg. per kilogram) before and again after treatment. Points lying between the broken lines illustrate absence of significant differences between the results of the two tests; points above this zone indicate a lower glycemic response to epinephrine after than before treatment; points below this zone indicate a greater glycemic response to epinephrine after than before treatment.

In five of the 21 studies made after treatment the amount of increase in blood sugar after injections of epinephrine was not significantly different from the result of the test done before treatment (chart 2). In eight of the 21 studies the rise after treatment was greater than that before treatment, and in eight others the reverse was true. The initial level had no effect on the direction of change.

COMMENT

Kinsell and associates [1] showed that after normal subjects have been prepared by the ingestion of very large amounts of carbohydrate for three days the increase in blood sugar after injection of epinephrine is between 40 and 100 mg. per 100 ml. of blood. Three fourths of the patients with mental disease in the present series

gave values in or above this range in the absence of this dietary preparation and in spite of having eaten poorly and lost between 5 and 30 per cent of their weight before admission. The data of earlier authors also showed normal or increased glycemic responses to the injection of epinephrine in patients with mental disease.[4] These findings indicate that an abnormal tendency toward glycogen formation, or impairment of glycogen breakdown, in the liver exists in patients with the psychoses studied or with severe neuroses, a phenomenon consistent with the effects of increased activity of adrenocortical sugar-active hormones. Decreased utilization of hepatic glycogen stores in spite of anorexia and the impaired ability to utilize glucose and lactate well known to occur in schizophrenia and depressions [3] imply that protein and fat must be used as sources of energy; these considerations serve to explain the creatinuria and ketonemia found in psychosis.

In vitro, the effect of the sugar-active adrenocortical hormones is to cause deposit of glycogen in the liver even if glucose is not available, protein then becoming the substrate.[6] However, the amount of carbohydrate available is important in determining the amount of glycogen deposited,[7] so that the diet, however meager prior to the test, might be expected to influence the result. In addition, it is possible that fatty changes in the liver, consequent to severe nutritional deficiency, might also give a low result. It is therefore remarkable that so many high values were found before treatment.

In spite of improvement in nutritional status and gain in weight of between 10 and 40 pounds (4.5 and 18.1 Kg.) in all the patients treated, in only eight of the 21 studies made after treatment was a greater rise in blood sugar found after injection of epinephrine, no change or a decrease occurring in the rest. Indeed, in five patients the values found after treatment were below the lower limit of normal for patients on a high carbohydrate intake. The validity of results obtained with the test several days to a week after the end of shock treatment has not been established, so that these findings cannot be interpreted with certainty. Borenz and associates [4j] also found variable changes in the glycemic response to epinephrine after treatment; their values are difficult to compare with those obtained in the present study because they did the epinephrine test two hours after giving 0.1 unit of insulin per kilogram of body weight, a procedure itself likely to cause changes in sugar metabolism. Additional work is now in progress bearing on the validity of the test after shock treatments.

SUMMARY AND CONCLUSIONS

The rise in blood sugar after the injection of epinephrine was larger than would be expected in most patients with schizophrenia, manic-depressive psychoses and severe neuroses. This finding suggests an increased tendency to form hepatic glycogen in spite of starvation and malnutrition. After a course of insulin treatments (patient ambulatory) or of electric shock therapy, the results of the test were variable; the validity of the test at such times has not been established.

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7. Ingle, D. W.; Prestrud, M. C.; Li, C. H., and Evans, H. M.: The Relationship of Diet to the Effect of Adrenocorticotropic Hormone upon Urinary Nitrogen, Glucose and Electrolytes, Endocrinology **41**:170, 1947. Ashby, W. R.: Adrenal Cortical Steroids and the Metabolism of Glutamic Acid in Gluconeogenesis, J. Ment. Sc. **95**:153, 1949.

TRAUMATIC NEUROSIS, COMPENSATION NEUROSIS OR ATTITUDINAL PATHOSIS?

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TRAUMATIC neuroses have long been a troublesome medical and forensic problem. When the matter of financial compensation (gain) is involved, the question immediately arises: Are we dealing with a compensation neurosis? Is there a difference between a traumatic neurosis and a compensation neurosis? If so, what is that difference? In other words, when a person exhibits a set of functional nervous symptoms after an accidental injury and the person responsible for the injury has a financial obligation to the victim of the accident, how can one assess the respective roles played by emotional trauma per se, on the one hand, and by the unconscious desire for financial remuneration, on the other? In order not to confuse the issues involved in this question, I shall eliminate from this discussion any consideration of malingering, i. e., conscious simulation of symptoms for the sole purpose of gaining financial or other forms of profit. However, even with this limitation, one is still left with a group of cases which neither fall into the group of traumatic neuroses nor have the characteristics of a compensation neurosis. I shall call these conditions the "attitudinal pathoses." [1]

Since World War II the world has been neurosis minded. The literature on traumatic neurosis and its allied conditions is encyclopedic, and much of the terminology is confusing, unsatisfactory and inadequate. For this reason, I shall attempt to clarify our ideas of traumatic neurosis and compensation neurosis and then add what I believe to be a new operational concept that is both necessary and useful.

TRAUMATIC NEUROSIS

Kardiner [2] states that the importance of traumatic neurosis is due not only to the severe incapacities which result from it, but also to the many and complicated forensic problems which it brings in its wake. The chief of these problems is that of compensation. The essence of the traumatic neurosis is not an organic lesion but a failure in the victim's total possibilities for adaptation. In other words, the traumatic neurosis is "psychogenic," i. e., the resultant of conflicting forces or drives within the personality structure of the individual. Dynamically, the traumatic neurosis is a reaction not specifically related to the type of injury which caused it. Therefore, it is misleading to designate a neurosis according to the provoking situ-

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ation, i. e., "railroad spine," "shell shock," "autonomic imbalance" or "lightning neurosis." The violence and nature of the accident merely color the intensity of the neurosis rather than determine its essential character. The accident is a "symbolic" stimulus. Rapoport [3] points out that the response to a symbolic stimulus is, in general, independent of the stimulus. A primitive organism responds to a stimulus according to its intensity. That is, hot water applied to the foot of a frog causes the frog to withdraw the foot. The hotter the water, the more rapidly the foot is withdrawn. But, according to Rapoport, the intensity of a symbolic stimulus has no significant effect on the response. If one whispers to a person the words, "There is a rattlesnake right behind you," he will take as drastic an action as he would if the words were shouted. The effect of a direct stimulus can be compared to that of a stretched bowstring; the more the string is stretched, the farther the arrow flies. On the other hand, the effect of a symbolic stimulus is like that of a released trigger. It does not matter how hard the trigger is pressed: It is not the trigger that sends the bullet on its way; it is the powder charge inside the gun. Similarly, the action of a symbolic stimulus is not inherent in the stimulus itself; it is the reaction that the stimulus sets off inside the recipient.

It must be emphasized that in true traumatic neurosis the indemnification issue does not in any way create the disorder. The neurosis is not dependent on the unconscious use of the accident to solve difficulties that have nothing to do with the trauma. Davidson [4] believes that traumatic neurosis is an effort to solve a conflict between adventure and security. On the one hand, the patient desires the security offered by the secondary gains he derives from his neurosis (freedom from responsibility, attention, sympathy and various other satisfactions of infantile strivings); on the other hand, he thirsts for the adventure of being independent, productive, mature and the recipient of community approval. Anxiety develops when the patient is immobilized. Narcissistic regression takes place, and object relationships are replaced by narcissism, characterized by faulty cathexis of these object relationships and a return to an earlier narcissistic level of functioning. A withdrawal of object cathexis changes the mental economics, so that amounts of libido that were normally connected with the ideas of objects now intensify all ideas concerning one's own organs.[5] If a collapse of the reality-testing functions of the ego supervenes, whether owing to somatic or other causes, a condition simulating a schizophrenic episode may develop. In later life some persons react to any narcissistic hurt in the same way that they attempted to react to their first narcissistic hurt, i. e., to the realization that they are not omnipotent. This regression actually is a means of defense against fears and conflicts arising at the id level and concerning the infantile sexuality. The ego becomes passive and weak but, in the neuroses, not disorganized.

In my experience, true traumatic neurosis in the sense that it has just been characterized is a relatively uncommon condition. Practically all the patients whom I have seen with this disorder have been people for whom there was no prospect of financial return on their illness or whose litigation and indemnification had long

3. Rapoport, A.: Science and the Goals of Man, New York, Harper & Brothers, 1950.

4. Davidson, H. A.: Neurosis and Malingering, Am. J. M. Jurisp. **2**:94 (Feb.) 1939.

5. Fenichel, O.: The Psychoanalytic Theory of the Neurosis, New York, W. W. Norton & Company, Inc., 1945.

since been settled. Wechsler [6] made the diagnosis of traumatic neurosis in but five of 100 cases of head injury which he studied. However, some of these 100 persons had organic cerebral damage. Wechsler points out that the terms "traumatic neurosis" and "traumatic hysteria" are not to be used synonymously or interchangeably. With him, I believe that they are two distinct entities. Dynamically speaking, traumatic neurosis is a narcissistic regression in which the ego instincts, rather than the psychosexual instincts, are affected. Traumatic hysteria is a conversion hysteria and is not to be considered as part of the present discussion. Persons with genuine traumatic neuroses are those who have previously been well adjusted and, after a relatively mild, or even trifling, injury to the head or body manifest a set of variegated symptoms involving their psychic living in all its implications. In traumatic neurosis the reference is chiefly to body ego, and the reaction is characterized by the absence of displacement phenomena, which make up the bulk of the material in the conversion hysterias.

COMPENSATION NEUROSIS

While the sufferer from a compensation neurosis believes consciously that he is sick, a careful psychological examination discloses a volitional factor operating in the disability, and also indicates that the neurosis was created in part by a subjective conviction on the part of the patient that he has been in a compensative accident. In my experience, the great majority of the postaccident neuroses are of this type. This disorder is precipitated by certain environmental factors acting on personality defects. One of these factors is the prospect of indemnification. If these environmental factors are so modified (denial of compensation) that they cannot favor a voluntary reaction, the psychological reaction will change in the direction of adjustment. This is the principal reason that the symptoms shown by patients suffering from compensation neuroses (frequently miscalled "traumatic neuroses") almost invariably disappear within a short time after a legal and financial settlement has been made. I believe that it is unwise to continue the payment of weekly disability benefits to a sufferer from a compensation neurosis. It is much better to effect lump sum settlement, for in this way the disability is not converted into a gainful occupation. However, in spite of the existence of a volitional factor in compensation neurosis, we must avoid the mistake of considering the whole reaction as malingering, and we must not evaluate the condition from this point of view. I feel that people suffering from a compensation neurosis are just as sincere in their belief that they are sick as are those who have a traumatic neurosis. The volitional integrant is at least foreconscious, and it may even be unconscious. However, it is suppressed by fear, indignation and resentment.

Fear is engendered in many cases by injudicious remarks on the part of physicians and attendants who are with the patient at the time of, or shortly after, the accident. The accident itself may be a life-threatening experience, and the effect of this experience is enhanced if the patient hears some person remark, "My but that's a tremendous laceration," or "He's lucky his brains aren't bashed in." This type of suggestion seeps into the patient's unconscious mind and perpetuates the anxiety which helps to mask the desire for financial gain.

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6. Wechsler, I.: Trauma and the Nervous System, with Special Reference to Head Injuries and a Classification of Post-Traumatic Syndromes (Analysis of 100 Cases), J. A. M. A. **104**:519 (Feb. 16) 1935.

Indignation and resentment result from skepticism on the part of the "company doctor," the foreman, the nurse at the first aid station or the insurance adjuster. These people sometimes are apt to question the genuineness of the patient's symptoms and the validity of his claim that he was injured while at work. Resentment not only accentuates the symptoms, but prolongs them and presents a serious obstacle to rehabilitation.

Forel cites Kretschmer [7] as stating:

> Resentment is the complex attitude of mind of those who, in fact, have suffered injustice, or deem themselves to have been injured. It is to see life in perspective from below; it is a constant gnawing feeling of rebellion, the many-sided attitude of the weak in relation to the powerful, of the poor toward the rich, of the sick, the degenerate, and the disintegrating toward health and youth—in a word, the mental attitude of all malcontents who are constantly ready, in their "life envy," to revenge themselves on or to continue in their state of malicious resentment.

With resentment goes a wish for atonement from the privileged.[7] One finds types of resentment in every class of people. The workman who has been injured while in the employ of a corporation, the person who feels that the insurance company (symbolizing the powerful "upper class") is in some way responsible for his woes or he who believes that he should be indemnified for what has happened to him are no exceptions. Their resentment creates either a conscious or an unconscious desire for revenge, and one way of getting revenge is either to remain disabled for a long time or to make the object of the resentment pay out in dollars. Like all dynamic feelings, resentment tends to expand. Therefore, in any postaccident neurosis it is desirable to have all questions involving compensation settled as soon as possible after the determination has been made of the extent of the patient's disability.

There is no clearcut dividing line between the true traumatic neuroses and the compensation neuroses. Rather, the features differentiating these two conditions form a spectrum. We cannot adhere to the two-valued "either-or" orientation. A condition need not be *either* a traumatic neurosis *or* a compensation neurosis, for there are situations in which the dynamic factors of both entities are operating simultaneously. In our thinking, we must avoid the semantic error of adhering to a strictly two-valued orientation, and we should recognize the existence of the spectrum extending between the two absolutes.

One of the most difficult jobs for the medicolegal expert is the determination of how much of the postaccident syndrome is due to a true traumatic neurosis (narcissistic regression) and how much to a compensation neurosis (foreconscious or unconscious desire for gain). The Rorschach test may be one method of arriving at a proper evaluation of the relative strengths of the different components. Although I do not have any statistical information regarding the Rorschach findings in true traumatic neuroses as compared with the findings in compensation neuroses, it is possible to evaluate by the Rorschach method the degree of regression and the preponderance of or lack of a true neurotic reaction. In fact, it should be possible to quantify these findings within certain limits. Time and space do not permit a discussion of the implications of the Rorschach test in connection with traumatic neurosis and compensation neurosis, but such workers as Beck [8] have gone a long

7. Forel, O. L.: Resentment: An Obstacle to Reëducation, Ment. Hyg. **33**:177 (April) 1949.

8. Beck, S. J.: Personal communications to the author.

way toward arriving at statistical evaluations of Rorschach responses and assessing their dynamic importance in the personality configuration. I have submitted the results of Rorschach testing as part of the basis of my opinion in several medicolegal cases, and in some courts the testimony regarding Rorschach findings was given considerable weight. Of course, it must be remembered that the Rorschach test is a highly complicated and very sensitive one. It also must be remembered that there are many persons with insufficient training and little experience who are doing Rorschach tests. Further, there is still a great deal to be learned about the Rorschach test. However, on the basis of sound statistical work, such as that done by Beck and his co-workers, a number of valid concepts have emerged, and these concepts are experimentally verifiable. Although there still is much work to be done, one feels that, in view of the mess in which our medicolegal testimony regarding traumatic neuroses, etc., finds itself, anything which will clarify our thinking and quantify the operational effect of various dynamic factors will be very worth while.

ATTITUDINAL PATHOSES

For a long time I have suspected that there exists a large number of persons who suffer from postaccident symptoms and who have neither a traumatic neurosis nor a compensation neurosis. They do not have the reactions of a true neurosis, and, at the same time, the question of compensation is not involved. Therefore, it was with great interest that I read the report of Dr. Frederick Thorne [1] regarding the attitudinal pathoses. In discussing this concept, I shall quote Dr. Thorne freely, and occasionally verbatim. To begin with the functional postaccident syndromes are forms of personality disorders. In many of these disorders, morbid attitudes held by the patient are the primary etiologic factor. The psychiatric nomenclatures and classifications in use at present are unsatisfactory in furnishing a valid differentiation between a large number of syndromes of maladjustment encountered in "normal" people and also in those persons living on the borderline between mental health and mental disorder. In other words, to return to the semantic fallacy of adhering to a two-valued orientation, it is not necessary that a person be either neurotic or not neurotic. He may be suffering from a group of symptoms which had their inception after a physical accident, and yet he is not sick according to present medical standards. He may present a syndrome of maladjustment which is related to a morbid attitude.

> The term attitude is defined as implying a subject-object relationship involving more or less enduring states of readiness, having affective properties of varying degrees, varying in the number and range of stimuli to which it is referred, and being formed by experiences in relation to persons, objects, and values. Included under attitudes are the ideas, opinions, beliefs, prejudices, values, etc., which a person acquires through experience and which are dynamically organized by the individual field of forces operant at any given moment.[1]

The formative notion of "morbidity" is used in a completely relative sense, and it refers to attitudes that are so deviant or atypical as to constitute etiologic factors in maladjustment.

Note already how many familiar postaccident syndromes can be accounted for on the basis of morbid attitudes! But let us proceed.

Attitudes occur in constellations, i. e., groupings or configurations of attitudes which have a common referent. Every constellation has a nucleus of central

attitudes which is called the nuclear core attitude. When constellations of attitudes become sufficiently discrete and complex to form clinically recognizable syndromes, they are called complexes, and suitable descriptive names are attached, e. g., inferiority complex, superiority complex and persecution complex.

Implicit in all forms of therapy is the recognition of the need to modify morbid attitudes which are etiologic factors in maladjustment and mental disorder. Lewy [9] speaks of attitude therapy, which aims to modify psychiatric problems in children by changing the attitudes of their parents. All psychotherapists are aware of the important part played by faulty attitudes in adult maladjustment, and, in their own parlance, they frequently use the term "basic attitude." In the frame of reference used here, this would be called the "nuclear core attitude."

Attitudinal maladjustment may be of any degree of complexity and malignancy, and they range from single relatively benign pathological attitudes ("I won't eat meat"; "All Jews are dishonest") to the relatively malignant constellations of pathological attitudes, which Thorne has called attitudinal pathoses. The reason for introducing a new classification is that the pattern of maladjustment here described cannot be logically subsumed under the classic syndromes. An attitudinal pathosis is a global disorder, which involves not only the primary pathological attitudes but also the secondary personality and environmental reactions. In this way it differs from a pathological attitude which is merely one of the basic elements.

Although modern field theory in psychology has given us insight into the global or molar aspects of personality, it is also important to adopt a molecular approach in analyzing attitudinal complexes or neurotic structures. Attitudes are organized according to the principles of unification and self consistency, and every constellation seems to be organized about one or more core attitudes. Given the nuclear core attitude—"I have been injured and cannot work"—one accepts accretions to this central attitude according to the principles of unification and self consistency. Therefore, one will develop a constellation composed only of attitudes which are consistent with the nuclear core attitude. Whether or not there is anything physically wrong need not enter into the picture. Assume that a person has sustained a minor injury and that he has completely recovered from the physical effects of this injury. If he adopts as his nuclear attitude the idea that because he has been hurt he is unable to work, he is going to interpret reality according only to attitudes which are consistent with his basic, or core, attitude. As will be shown later, this is not a true neurosis, nor does the question of indemnification necessarily enter. Suppose that an injured workman has as his nuclear core the attitude that he has been dealt with unfairly. Again, accretions to this core will consist only of attitudes which are consistent with the basic attitude, and any attitudes which are not consistent will be rejected, e. g., that the company is trying to help him, that they are interested in his rehabilitation or that he will get his old job back without prejudice. Finally, suppose an injured worker has a nuclear attitude that because he has been injured while at work he is entitled to some special consideration, not necessarily financial remuneration. He refuses to go

9. Lewy, E.: Contribution to the Problem of Compensation Neuroses, Bull. Menninger Clin. 4:88 (May) 1940.

back to his old work, and he insists on being given lighter work with shorter hours at the same pay he received before he was hurt. He convinces the shop steward and then his local union that such is the case. The union accepts the same core attitude and, in turn, develops a constellation only of attitudes which are consistent with the central idea. Attitudes favoring compromise are rejected, and the dispute ends in a prolonged strike against the company. I have seen this happen. It cannot be said that the injured workman is in this case a malingerer; neither was he suffering from any kind of a neurosis—traumatic or compensation. He and the union were suffering from an attitudinal pathosis which was a global reaction involving not only the original pathological attitude but also the secondary personality and environmental reactions.

Theoretically, an ideally healthy personality, or even an ideally healthy society, would be one "in which all attitudes were logically or scientifically valid, internally consistent and positive."[1] When attitudes are invalid, inconsistent or ambivalent, and when they negate the principles of unification and self consistency, conflict and maladjustment occur. In favorable circumstances, it is possible to bring about any alteration of personality (either in the direction of health or in that of maladjustment) by the manipulation of appropriate attitudes. Thorne refers to the studies of the inmates of concentration camps, of the conversion of alcoholics, prostitutes and criminals under the conditions of controlled attitude modification as indicating the possibilities which exist. I feel that the same can be done for a man or woman whose postaccident morbidity is the result of faulty basic attitudes. Thorne says that, while under optimum conditions these changes can be achieved nondirectively, they can be achieved more consistently and in greater magnitude with directive methods. Since attitudes are learned, they are subject to change by unlearning and relearning. They obey the laws of the psychology of learning.

Attitudinal pathoses differ from the neuroses in that they are more limited, circumscribed, conscious, voluntary and intellectual than the psychoneuroses. The traumatic neuroses and, to a certain extent, the compensation neuroses are related in a large measure to unconscious or foreconscious, involuntary processes, which run on automatically largely outside conscious control. As Thorne puts it, "The neurotic reaction is typically more severe and disabling to the total personality; it is a more global disorder." However, if the affective life is deeply involved, it is conceivable that attitudinal pathoses may merge into full-blown neurotic reactions. That is why the elements of resentment, hostility and anxiety must be recognized and managed. In the attitudinal pathoses the person consciously behaves in a certain way because he believes that he is "right." In a neurosis he behaves in a certain way because of complexes which he is unable to control, even though he realizes that his behavior is maladjusted and illogical. In other words, the neurotic is motivated by hidden conflicts, narcissistic regression, free floating anxiety and hypochondriacal preoccupations which he recognizes as being illogical, while the sufferer from an attitudinal pathosis is motivated by constellations of ideas of which he is consciously aware and which he thinks are logical. The neurotic is cognizant of the dissonance between his feelings and the reality of his situation, while a person with an attitudinal pathosis believes that there is a consonance between what he believes and what he feels—i. e., "I was injured during the course of my employment; there are compensation laws; I am entitled to com-

pensation; I have a backache which came on after I was injured; therefore, I cannot go back to work until I have received what I believe to be just compensation for my 'injury.'" This is the nuclear core attitude around which the worker builds constellations of other attitudes, all consistent and unified with the core. Add to this resentment, hostility and anxiety, and it is not difficult to understand how the patient may be driven into a regressive neurosis, even after the question of indemnification has been settled. If by the time the patient goes into his neurosis the indemnification question has not been settled, the condition may lie somewhere in the spectrum between true traumatic neurosis and compensation neurosis. Every problem must be dealt with individually. We cannot make any mass generalizations one way or another, and the appraisal of a given case will depend on the skill, experience and insight of the psychiatrist called on to evaluate the situation.

FORENSIC IMPLICATIONS

When applied to personal injury suits, the concept of attitudinal pathosis may have rather broad forensic implications. While the courts recognize traumatic and even compensation neuroses as compensable disorders, there may be some question as to whether a person with a postaccident attitudinal pathosis is entitled to the same treatment and the same indemnification as is the neurotic. While some compensation statutes hold that any state of mind, regardless of its nature, having a causal connection with an industrial accident and resulting in symptoms which are disabling is compensable, there is some question in my mind as to whether the same theory should be applied to the attitudinal pathoses. Although the sufferer from an attitudinal pathosis is honest and sincere in his convictions, and although he has no conscious intent to fake or to commit fraud, the factor of volition is so much more dominant in the attitudinal pathoses that the legal philosophy of the postaccident syndromes probably should be modified with this in mind. If we psychiatrists who deal with large numbers of postaccident disabilities will bear in mind the possible existence of an attitudinal pathosis, it might be that in a few years we shall have a sufficient number of observations and reports to enable us to make some recommendations to our colleagues in the field of medical jurisprudence.

SUMMARY AND CONCLUSIONS

Post-traumatic neurosis is a relatively uncommon condition, and it represents a narcissistic regression. In pure form it is not related to the question of indemnification.

Compensation neurosis is a true neurosis, but one in which the unconscious desire for gain plays an important part. It is by far the commonest of all the functional postaccident psychiatric syndromes.

Attitudinal pathosis is a form of personality disorder in which the patient consciously behaves in a certain way because he thinks he is "right." He is not malingering. His motivation is a constellation of attitudes built around a nuclear core attitude which is faulty. Emotivity is disturbed only secondarily, and a feeling of resentment is the principal reaction.

Inclusion of the attitudinal pathoses among the postaccident syndromes may reflect in modifications of some of the present concepts in the field of medical jurisprudence.

ABSTRACT OF DISCUSSION

DR. GEORGE N. RAINES, Bethesda, Md.: Dr. Kamman has given us a good idea of at least three of the fashions in which a personality responds to injury.

The differential diagnosis of the traumatic neurosis and the compensation neurosis is, indeed, difficult, for the very reasons that Dr. Kamman has mentioned, namely, that one deals with the spectrum rather than with the ends of the spectrum. It becomes essentially a matter of weighing the components of an illness rather than of differentiating two clinical entities. Traumatic neurosis, to us, has usually meant the result of a severe, acute fear or of repeated small (if you will) chronic fears, such as are seen in combat fatigue. Ordinarily, the result of this is the classic picture of startle reaction, nightmares, and the like, fading off into the so-called compensation neurosis, which, interestingly, was not very common in the military service (at least, in the Navy). We did see it. Compensation there ordinarily was in the nature of discharge from the service. Officers were entitled to retirement by virtue of physical disability, and among them the frequency of compensation neurosis, as described by Dr. Kamman, increased.

I find some difficulty with the term attitudinal pathosis. Psychiatry already uses more terms than most of us can understand or define. The introduction of a term of this type is discomfitting to one who has worked at revising psychiatric nomenclature. It is difficult to define the attitudinal pathoses. The author of the term itself, Dr. Thorne, points out that it is primarily a personality disorder. It is now recognized that most mental illnesses are the result of the effort of the personality to adjust to environmental pressures with what resources it has at hand. Consequently, when one coins such a term and immediately recognizes it as a symptomatic diagnosis, one is going around in circles. The name, as defined, has no more specificity than the diagnosis of alcoholism, which can be symptomatic of a number of disorders.

However, as usual, the name is coined because of the need for a name. The same need led, toward the end of the war, to the Army's revising its nomenclature entirely. Psychiatric nomenclature has been written by psychiatrists, but many of these patients have not been seen by them. Patients of this type go to the neurologist, who limits himself to a diagnosis of organic or nonorganic disease and labels it neurosis; or they go to a general physician. Consequently, the large body of psychiatrists have seen little or no need for the provision of diagnostic categories covering this particular field.

In the military service, we were forced into revision. In 1944, for example, roughly 3,000 to 4,000 patients were discharged from the United States Naval Hospital in Portsmouth, Va., 40 per cent of whom fell into this category of "attitudinal pathosis." Our only available diagnosis was "constitutional psychopathic state." The minute we tagged a relatively successful young man (I mean in his own terms) with such a diagnosis the family descended on us in rage. The Army had so much of this pressure that they revised their nomenclature.

When a person is injured, he attaches to that injury whatever use he may have for the injury. For example, certain schizoid personalities hung onto injuries for compensable purposes (compensation being discharge) because of some intolerable pressure aboard ship. The injury was used to allow them to escape the particular situation honorably.

It is for this reason that I think this symptomatic diagnosis is somewhat superficial in conducting the patient's therapy. When a person is said to take a "nuclear core attitude," the question immediately follows, "Why?," for the assumption of that attitude itself serves some purpose to that patient.

I believe, however, that Dr. Kamman has brought to our attention an important and much needed formulation, namely, that the patient must be considered in terms of an individual struggling with his environment with whatever resources he can lay his hands on.

DR. MORRIS J. TISSENBAUM, Brooklyn: I should like to comment on several items in Dr. Kamman's paper which pertain to the Veterans Administration. Compensation for disabilities is an important feature of Veterans Administration work. Dr. Kamman speaks of factors of indignation and resentment in connection with compensation neuroses and stresses that these factors prolong disabilities and make treatment difficult. With this concept I agree, and I should like to stress that many veteran patients have carried with them the same factors

of resentment, hostility and indignation which they had during military service toward officers and the Army and Navy in general. The physicians and other personnel of the Veterans Administration have taken the place of officers, and the Veterans Administration itself appears to have taken the place of the Army or Navy as the butt of their resentment. It is my feeling that these attitudes go back to early developmental factors and are tied to basic attitudes, especially toward the father figure and, subsequently, anything which represents authority. This they have brought into the service and, ultimately, the Veterans Administration.

In these situations, where can one draw the line between an attitudinal pathosis and a compensation neurosis? A persistent and fixed attitude which will affect a man's behavior to such a degree that it becomes a personality disorder is an illness and must derive from a deep-seated pathologic state. Are there not real neurotic, even psychotic, patterns in these attitudinal pathoses?

In a survey in the clinic in Brooklyn, involving 329 veterans with a diagnosis of post-traumatic encephalopathy, it was interesting that the majority presented purely psychiatric symptoms, with no evidence of organic neurologic change. All these men are collecting compensation from the Veterans Administration. A review of these cases seemed inconclusive as to the role of compensation. It is my feeling that these patients respond to treatment because they have neurotic phenomena amenable to therapy. Where there is an element of secondary gain involving compensation, it is handled therapeutically, just as is any other element of secondary gain found in a neurotic syndrome.

It is my feeling that there is no proof that the so-called secondary gain of a pension plays a delaying effect in psychiatric rehabilitation, even though this concept has been stressed frequently. It is part of the entire picture which has to be treated, like any other symptom.

Dr. Gordon R. Kamman, St. Paul: I think there is a difference between the war neuroses and the neuroses one sees in industrial situations. In civilian life, the true traumatic neurosis is encountered rarely, and only in cases in which indemnification is not involved. For instance, I saw a patient two weeks ago who had fractured her ankle in her own home a year previously, and she still had a full-blown, typical neurosis, which we thought had been initiated by the trauma, the loss of object relationships, the centering on herself and the disturbances of emotivity.

I have seen, as I said, comparatively few traumatic neuroses. Another woman was in an automobile accident, with no question of compensation involved. The accident was in Illinois; she went back to her home in St. Paul and two years afterward was in a hypochondriacal state, which I felt was directly related to the traumatic neurosis. Indemnification was not involved in this case.

I agree with Dr. Raines that "name psychiatry" is something to be deplored, and I am firm in my belief and teaching that we should not be interested in labels. We are interested in processes and in what is going on and in what can be done. However, we have, for purposes of the forensic implications, to use names.

Would you say that a person is neurotic or psychopathic who has as a nuclear core attitude the idea that the earth is flat; in the interest of consistency, accepts only ideas consistent with that, and, as a result, has difficulty in understanding what Magellan did? I think he would have an attitudinal pathosis.

I know of rather odd religious sects, with strange beliefs, strange prohibitions and taboos. Would you call their followers neurotic? Would you call them psychopaths?

I know of certain people who have nuclear core attitudes regarding labor unions which are not in agreement with the commonly accepted attitudes. They have increments to these core attitudes which motivate their total behavior. Would you call these neurotic? No.

By the same token, people who are injured and who have the nuclear core attitude, "Well, my employers carry compensation insurance and I have been injured," do not necessarily have to have a neurosis, nor are they malingerers. The physical effects of the injury have disappeared, although the patient does not admit it. Their doctor has them continue to come in and injects thiamine chloride for a period of 18 months, and the constellation of attitudes becomes set. However, it is not a true neurosis.

A woman I saw recently had an idea that because she had a backache after her injury she had to have some compensation. She did not have any signs of a neurosis; there was no disturbance of her emotivity, and there was none of the regression we see in neurosis. Yet she had a condition for which she was demanding compensation. Her case was brought before the Industrial Commission. I did not want to say that she was a malingerer, because she was sincere in her belief. At the same time, I could not say she was neurotic.

That is the reason we should use this concept of attitudinal pathosis, although I do believe, with Dr. Raines, that "name psychiatry" is something to be deplored and something to be avoided whenever possible.

Why should people have such morbid attitudes? Why does a person have to believe that the earth is flat or that he cannot kill a living thing? I think the anthropologists will have to help us out in some of those problems.

I know I took a chance when I suggested the introduction of another name, but I still feel there is a place for it.

A DYNAMIC FACTOR CORRELATED WITH THE PROGNOSIS IN PARANOID SCHIZOPHRENIA

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WITHIN the diagnostic category of paranoid schizophrenia, one may distinguish two clinically dissimilar varieties of reaction to projected judgments of guilt. The patients in one of these groups passively accept their projected judgments of guilt. For example, one such patient had an auditory hallucination in which he heard a voice saying, "Get that bum; he's no good." The subsequent reactions of this patient indicate that he passively accepted this projected judgment of guilt. Instead of stopping to refute the projected accusation, he fled, apparently in terror, plunging headlong through the screen door of the barracks in which he was living. In order to distinguish such patients from other paranoid schizophrenic patients who do not passively accept any judgments of guilt, even their own projections if they have them, Molholm [1] has suggested that these two types of paranoid schizophrenia can be described as the passive, compliant type and the active, defiant type. It should be noted that Molholm became interested in the passive, compliant type of paranoid schizophrenia when he found that all recently hospitalized schizophrenic patients who did relatively poorly on the Kohs Block Design test had usually begun their psychosis by having a projected judgment of guilt which they passively accepted.

In 1945, Molholm and I suspected that the compliant type of paranoid schizophrenia was associated with a better prognosis than the defiant type. This idea was based on clinical impressions and certain theoretical formulations. The conceptual aspects of this problem will not be dealt with here, since they will be published separately by Molholm. The purpose of this report is to present the results of an investigation in which the prognosis for the defiant and compliant types of paranoid schizophrenia was studied.

PRESENT STUDY

The medical records of 69 patients with the diagnosis of "paranoid schizophrenia" were selected at random. These patients were then separated into two groups, depending on whether they defiantly resisted or compliantly accepted

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1. Molholm, H. B.: Personal communication to the author.

accusations, including their own projected judgments of guilt. Data pertaining to type of treatment and disposition of patients were collected. The following relations were found:

Of the 69 patients randomly selected for the diagnosis of paranoid schizophrenia, 50 (72 per cent) were classified as defiant and 19 (28 per cent) as compliant. Fifty (72 per cent) of the total series of 69 patients were unimproved by treatment (of all kinds), whereas 19 (28 per cent) were sufficiently improved to be discharged with permission.

Of the 50 defiant patients with paranoid schizophrenia, only 8 (16 per cent) could be discharged, 42 (84 per cent) being unimproved by treatment (of all kinds). Of the 19 compliant patients, 11 (58 per cent) were discharged after treatment (of all kinds), 8 (42 per cent) being unimproved.

Of the total series of 69 patients, 29 (42 per cent) received electric shock as the principal form of treatment. Twenty-one (72 per cent) of these were defiant, and 8 (28 per cent) were compliant. This is the same ratio of defiant to compliant patients as that in the total series of 69 patients.

Of the 29 patients receiving electric shock treatment, 10 (35 per cent) were discharged, and 19 (65 per cent) were unimproved. This is a 7 per cent better result than the series achieved as a whole, and 13 per cent better than that for patients not receiving electric shock therapy (remission in 9, or 22 per cent; no improvement in 31, or 78 per cent).

Of the 21 electric shock-treated defiant patients, 4 (19 per cent) were discharged, and 17 (81 per cent) were unimproved. This result is only 5 per cent better than that achieved for the 29 defiant patients not receiving electric shock therapy (remission in 4, or 14 per cent; no improvement in 25, or 86 per cent).

Of the eight electric shock-treated compliant patients, 6 (75 per cent) were discharged, and 2 (25 per cent) were unimproved. This result is 30 per cent better than the results achieved for the 11 compliant patients not receiving electric shock therapy (remission in 5, or 45 per cent; no improvement in 6, or 55 per cent).

Statistical evaluation of the significance of the differences between these various proportions was carried out, using the formula:

$$\sigma_{\bar{x}_1 - \bar{x}_2} = \sqrt{\frac{P_1 \times q_1}{n_1 - 1} + \frac{P_2 \times q_2}{n_2 - 1}}$$

This is a method for determining the standard error of the difference between two proportions. If two proportions differ by more than twice the value of the standard error of the difference, the difference would arise by chance, roughly, only once in 20 times. The following differences between the defiant and the compliant type were tested for statistical significance.

Fifty-eight per cent of the compliant and 16 per cent of the defiant patients were discharged as a result of treatment of all kinds. This is a difference of 42 per cent, which is more than three times the calculated standard error of the difference between the two proportions (12.6 per cent). This difference is, therefore, statistically significant and would indicate that the prognosis for the compliant type of paranoid schizophrenia is significantly better than that for the defiant type when treatments of all types are considered.

Comparing electric shock-treated patients only, 75 per cent of the compliant and 19 per cent of the defiant patients with paranoid schizophrenia achieved remission. This is a difference of 56 per cent, which is more than three times the calculated standard error of the difference between the two proportions (17.5 per cent) and is therefore statistically significant. This would indicate that when electric shock therapy is used as the principal form of treatment, the prognosis is significantly better for the compliant than for the defiant type of paranoid schizophrenia. Of the electric shock-treated defiant patients, 19 per cent achieved remission, whereas 14 per cent of the defiant patients not receiving electric shock therapy were discharged with remission. This is a difference of 5 per cent, which is not statistically significant, since it is less than twice the calculated standard error of the difference between the two proportions (10.7 per cent). This would suggest that electric shock therapy does not significantly improve the prognosis in the defiant type of paranoid schizophrenia.

Seventy-five per cent of the electric shock-treated compliant patients achieved remission, whereas 45 per cent of the compliant patients not receiving electric shock therapy were discharged. This is a difference of 30 per cent, which cannot be considered statistically significant, since the calculated standard error of the difference between the two proportions is 21.2 per cent. These findings, however, suggest a trend toward more improvement in the compliant than in the defiant patients as a result of electric shock therapy.

SUMMARY

According to a concept formulated by Molholm, patients with paranoid schizophrenia may be differentiated in terms of their reactions to accusations, including their own projected judgments of guilt. One group actively and defiantly resist such accusations and projections, whereas the other passively and compliantly accepts accusations, including their own projected judgments of guilt. This dynamic factor appears to be associated in a statistically significant way with the prognosis in paranoid schizophrenia. The following relations have been determined:

1. The defiant type of paranoid schizophrenia is approximately three times as common as the compliant type.

2. The prognosis for the compliant type of paranoid schizophrenia is significantly better than the prognosis for the defiant variety, regardless of the type of treatment employed.

3. Electric shock treatment does not significantly improve the prognosis in the defiant type of paranoid schizophrenia.

4. There is a suggestive, although statistically not significant, trend toward an improved prognosis in the compliant type of paranoid schizophrenia when electric shock therapy is employed.

5. In the defiant type of paranoid schizophrenia, approximately four of five patients will not achieve early remission, even if electric shock therapy is used. In the compliant type, approximately half of the patients achieve early remission regardless of the type of treatment employed, and as many as three fourths of these patients will recover if electric shock therapy is used.

THE PHENOMENON OF SENSORY DISPLACEMENT

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IN STUDIES employing the method of double simultaneous stimulation it was found that a subject may give one of three types of responses. Depending on various factors in the test situation, the subject may (*a*) perceive and correctly localize both stimuli (expected normal); (*b*) perceive and correctly localize one stimulus but not perceive the other, even though the latter was perceptible to the patient on single stimulation, and (*c*) perceive and correctly localize one stimulus and perceive but mislocalize the other. The first response needs no comment, for it is the expected normal. The second type of response was reported in a series of communications under the heading of the phenomenon of extinction.[1] The third type of response has been described to some extent in previous articles[2] under the heading of the phenomenon of displacement. However, the last phenomenon has not been stressed to any extent. It is the object of this communication to report observations on this type of perceptual reaction, with the special reference to the conditions under which the phenomenon of displacement can be found and the patterns it may assume.

MATERIAL

This study was carried out on patients with disease of the brain and spinal cord. The nature and location of the lesion and the degree of dysfunction of the nervous system were varied. Patients with psychosis or aphasia were included in the series, even though they exhibited difficulty in thinking and expression. In addition to the patient material, a series of normal adults and children were used as control subjects.

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2. (*a*) Bender, M. B.; Shapiro, M., and Schappell, A. W.: Observations on the Extinction Phenomenon in Hemiplegia, Tr. Am. Neurol. A. **74**:160-167, 1949; (*b*) Extinction Phenomenon in Hemiplegia, Arch. Neurol. & Psychiat. **62**:717-724 (Dec.) 1949. (*c*) Bender, M. B.; Fink, M., and Green, M.: Patterns in Perception on Simultaneous Tests of Face and Hands, Tr. Am. Neurol. A. **75**:250-252, 1950.

METHODS AND CONDITIONS OF TESTING

Cutaneous sensation was tested with methods of double simultaneous stimulation. The following conditions of testing were considered:

Factor of Locus of Stimulation.—In considering the cutaneous area to be tested, three principal methods of simultaneous stimulation were employed. 1. Homologous points on each side of the body, such as the hands, feet or both sides of the face were stimulated at the same time. 2. Nonhomologous regions, such as the right side of the face and the left hand or the left side of the face and the right foot, were simultaneously stimulated. 3. Two regions on one side of the body, such as the right side of the face and the right hand or the right breast and right foot, were simultaneously tested.

Factor of Attention.—The subject was asked to keep the eyes closed. The two stimuli were applied without warning and for a brief moment. It was then assumed that the subject was not paying attention to what was to be done. After removal of the stimuli, the subject was asked to report (*a*) what he felt, (*b*) how many sensations there were and (*c*) where they were perceived. If the response was other than the expected normal, i. e., stimuli perceived and correctly localized, the test was repeated. In instances in which repeated stimulations still resulted in defects in one of the perceptions the subject or patient was instructed to "pay attention" to the proposed stimulations or was informed that two stimulations would be applied.

Factor of Type of Stimulus.—Touch, pain (pinprick), vibration and, in a very few instances, temperature were the sensory modalities tested. Although in most cases the two stimuli were of the same modality, i. e., touch and touch, or pinprick and pinprick, in some instances they were mixed, namely, touch and pinprick, pinprick and vibration or rubbing and pinprick.

Factor of Stimulus Intensity.—Care was taken to insure approximate equality of the intensities of stimulation. Although algesiometers or other sensory measuring devices were not used, the manual pressures on application of the stimuli to the skin were judged to be equal. Occasionally the intensity of the stimulation was purposely altered; i. e., one of the two stimuli was deliberately increased in intensity.

Factor of Timing of Stimulations.—The stimuli were applied simultaneously in almost every instance. Occasionally, when the stimuli were applied a fraction of a second apart, the results were altered. As a rule the duration of the stimulation was a fraction of a second, but at times the stimuli were applied for a longer period. In some instances, the stimulation was repetitive, i. e. rubbing, tapping or interrupted pricking.

Factor of Difference in Degree of Sensibility of the Two Cutaneous Areas Simultaneously Tested.—In cases of a lesion of the brain or spinal cord with corresponding alteration in cutaneous sensation, the tests were carried out in the normal and hypesthetic regions. Stimulations were made simultaneously in a relatively normal and a hypesthetic area.

Factor of Drugs, Anesthesia and Electrostimulation of the Brain.—In a number of cases the patient was tested with double simultaneous stimulation under the following conditions: (*a*) after intravenous injections of subanesthetic doses of amobarbital sodium (sodium amytal®); (*b*) after general anesthesia, such as ether anesthesia, and (*c*) with repeated electric stimulations of the brain during electroshock therapy. In all these conditions the tests were performed soon after the subject was recovering from the anesthetic or convulsions.

RESULTS

Under the various conditions mentioned, tests with the methods of double simultaneous stimulation disclosed that some subjects responded by indicating that they perceived the two stimuli but mislocalized one of the sensations; i. e., they gave a type 3 response. This mislocalization was seldom haphazard. In general it was manifested in a definite direction. On the basis of numerous examinations, it was soon learned that the direction of the mislocalization could be predicted. Because the patient's "error" in localization usually showed direction and predictability, it was felt that the term "displacement" of sensation would be more suitable than

"mislocalization." Consequently, "phenomenon of displacement" will be the term used to indicate that under conditions of double simultaneous stimulation one of the sensations is "displaced" to another region of the sensory field.

Depending on the conditions of testing and the subject examined, there were three types of displacement of sensation. The displacement occurred away from the point of stimulation (*a*) in an ipsilateral direction, (*b*) in a contralateral direction or (*c*) out into extrapersonal space.

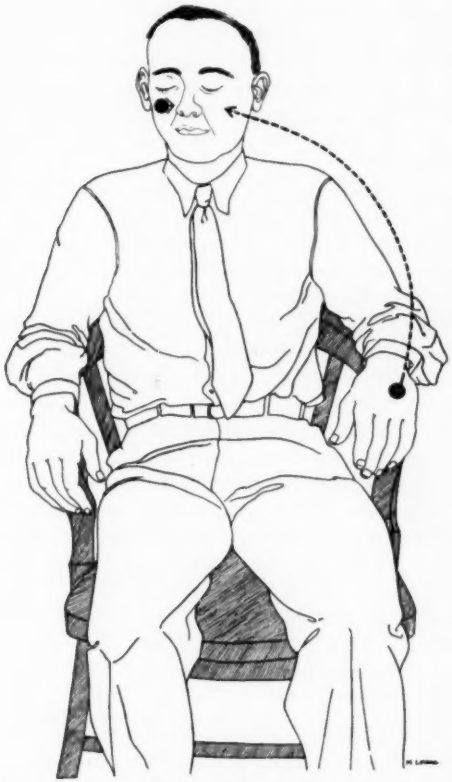

Fig. 1.—Direction of displacement in a normal subject sitting with eyes closed when tactile stimuli are applied simultaneously to the right side of the face and the left hand.

The solid black dot indicates the point of stimulation. The arrow indicates the patient's localization of percept. Two per cent of normal adults displaced the sensation from the hand to the face. No other type of displacement was noted.

The general incidence of displacement was low, averaging approximately 5 to 10 per cent. When displacement was present, the incidence also varied with the conditions of testing. Thus, on initial tests of simultaneous stimulation of the face and hand, the displacement phenomenon was found in 20 per cent of the patients with disease of the brain, in 2 per cent of the normal adult subjects and in 15 per

cent of normal children. However, on subsequent tests of the patients and the young children the incidence of displacement was raised, so that at one time or another displacement was found in a very high percentage. In other groups, in which the face and breast were tested, and in still other groups, in which the breast and hand were tested, the incidence of displacement also varied.

Whenever displacement occurred, the following reactions were noted: One of the two sensations was reported immediately and correctly; the other was perceived as vague. Often it was necessary to inquire of the subject whether another sensation was felt and, if so, where it was located. Thus, when the right side of the face and the left hand were stimulated simultaneously, the subject reported that he felt a sensation in the right side of the face; when he was asked, "Where else?" the answers were varied. Some of the normal adults would say, "I'm not sure whether there was another—is it here?" "I think it was here," pointing to the left side of the face. The patient with severe mental changes often denied the presence of the second stimulus.

The response varied not only with the individual subject but with the conditions of stimulation, as recorded above, namely, area of the body tested, attention, nature and intensity of stimulus and sensibility of the area tested. One significant factor was the location of the lesion and the degree of damage to the brain. For instance, in patients with diffuse disease of the brain and associated severe psychic disturbances, displacement was common on both sides of the body. However, in patients with localized disease of the brain, such as a lesion in one parietal region, displacement of sensation occurred only on the side contralateral to the lesion.

1. *Displacement in Patients with Severe Mental Symptoms Due to Disease of the Brain, but Without Apparent Sensory Defects on Routine Tests.*—The incidence of displacement in these patients was higher than in any other group. All types of displacement were found, namely, ipsilateral, contralateral to the point of stimulation and away from the body. When various combinations of the testing procedures were applied to different areas of the body, it was found that displacement occurred most frequently when the face and contralateral hand were simultaneously stimulated.

The phenomenon of displacement of sensation observed in patients with diffuse disease of the brain is described in the following case histories.

Case 1.—J. S., aged 48, the oldest of three brothers, manifested the syndrome of chorea with mental changes of varying degree. He gave a history that for approximately five years he had had "jerkings" in the feet and "cracking" sensations in the toes. There had been some difficulty in walking for three years, and for over one year prior to this study his movements had become clumsy and he had dropped objects held in his hands. For approximately two years he complained that his memory was poor. He had been forgetful, especially about little things. The father, who died in a psychiatric institution, had chorea and severe mental changes.

Psychiatrically, the patient was indifferent and placid and smiled frequently without provocation. His memory was defective, and there was a tendency to confabulate. Immediate recall was poor. There were errors in serial subtraction by 7's and in simple calculation, even though he was a college graduate. General information was poor. He showed difficulty in ability to imitate complex movements. He often failed in tests which involved more than one aspect of a situation.

The neurological examination disclosed distinct choreiform movements, most conspicuous in the fingers and toes. There was grimacing with bursts of laughter. Movements in the tongue were noted. There was generalized hyperreflexia, more pronounced on the left; a Babinski sign was elicited on the left, and its presence was suspected on the right.

The sensory examination with the customary method of single stimulation showed no apparent defect in any of the sensory modalities. However, with double simultaneous stimulation the patient showed defects in his responses, particularly when the face and hand were stimulated. There were three types of abnormal responses when the face and hand were touched at the same time: (*a*) He reported touch sensation in the face and not in the hand, even after being asked several times whether there was another sensation elsewhere; (*b*) he indicated touch sensation on both sides of the face when the face and the opposite hand were stimulated, and (*c*) he reported one sensation on the face while the one evoked from the hand was localized into space or onto a part of the body on which the hand was resting, such as the thigh. In a sitting position, with hands resting on his thighs and his eyes closed, the patient was touched or stroked with the examiner's finger in different regions of the body. The following tabulation indicates typical regions tested and the patient's responses.

Regions Touched	Response
Right side of face and left hand	Right side of face
Left side of face and right hand	Left side of face
Asked, "Anywhere else?"	"No"
Right side of face and left hand	Right side of face and pointing to left thigh
Left side of face and right hand	Both sides of face
Left hand	Correct
Right hand	Correct
Right side of face and left hand	Right side of face and left side of face
Right side of face and right hand	Right side of face and pointing into space above right thigh
Right side of face and left side of face	Correct

From this tabulation it is obvious that errors in perception were made in the hand and not in the face. Either there was no perception in the hand, or sensation was displaced from the hand to the face. In some instances there was displacement from the hand into space or onto the thigh on which his hand was resting. Repeated tests showed that these responses were present on numerous occasions, even after the subject was informed of his errors and instructed to pay attention. At times the patient gave a correct response, and only once out of 20 tests did he perceive the stimulus in the hand and not in the face. After he was told he was being stimulated in two places, one being the hand, he gave correct responses to double simultaneous stimulation. Then, after a variety of tests, examination with double simultaneous stimulation was repeated, and he again showed the above mentioned defects. When the patient was examined with simultaneous pinprick stimulations, the results obtained were similar, but the abnormal responses were not as frequent or as conspicuous as with touch stimulation.

CASE 2.—P. S., aged 44, the brother of J. S. (case 1), gave a history of fidgetiness, "nervousness" and defects in memory of 10 years' duration.

The neurological examination revealed nothing abnormal except for generalized hyperreflexia and choreiform movements involving all the limbs and the trunk, face and tongue. The chorea was not as pronounced as that elicited on examination of J. S.

Psychiatrically, there were severe defects in simple calculation. This patient had had one year of college education.

Examination with the method of double simultaneous stimulation, particularly of the face and hand, showed the same defects, as those noted in J. S., although they were not as marked or as frequent. There was (*a*) imperception in the hand, (*b*) displacement from the hand to the face or (*c*) displacement from the hand to the thigh on which the stimulated hand was resting.

Comment.—The clinical diagnosis in both cases was Huntington's chorea. It is significant that the defects in the face-hand test were present in both brothers but were more marked in the one with the severer mental changes. These errors

were manifest even though each patient was examined many times and was told to pay attention to the procedure. It is noteworthy that there were few, or no, sensory defects in the face but there were many errors in localization of the sensation evoked by a stimulus applied on the hand. Moreover, whenever displacement occurred, it was toward the face and not the hand; or in several instances it was away from the hand onto the thigh. All these observations indicate that the sensory

Fig. 2.—Direction of displacement in a patient sitting with eyes closed and hands resting on his knees. The solid dot indicates locus of stimulation, and the arrow, the point of perception in each test. Note the types of displacement from the hand, shown by dashes and arrows. *A* indicates displacement to the face, which is the most frequent type; *B*, displacement to the thigh, and *C*, displacement into extrapersonal space. Types *B* and *C* are rare and usually occur in patients with severe mental changes.

defects described show a pattern in which the face is most, and the hand is least, dominant.

More than 90 per cent of the patients with severe mental changes showed the phenomenon of displacement at one time or another. Despite the patient's apparent mental confusion, the displacement in sensation was usually in a definite direction

and showed an order of dominance of one area over another. On double simultaneous stimulation of the face and any other part of the body, the patient often correctly indicated perception in the face and made errors in reporting the other area stimulated. This was especially true if the hand was the other region tested. However, on double simultaneous stimulation of the hand and any other part of the body, the patient usually erred in localizing stimulation in the hand and was more or less correct in localization elsewhere, especially if the face was the other area simultaneously tested. The types of displacement observed were (*a*) ipsilateral, (*b*) contralateral and (*c*) into extrapersonal space. The last type of displacement was least common, while ipsilateral displacement was most frequent.

2. *Patients with Predominantly Unilateral Sensory Disturbances.*—Displacement was less frequent in patients who did not show severe mental changes. However, it was observed in cases of focal lesion of the brain with contralateral defects in sensation. The following case is illustrative.

CASE 3.—G. M., a woman aged 60, with a long history of hypertension, had a vascular accident in the right cerebral hemisphere. At the time of her admission to the hospital there was residual left hemiparesis. The psychiatric status showed some memory defects but no serious mental deficit. Cutaneous sensory examinations with methods of bilateral simultaneous stimulation revealed sensory extinction on the left side of the body, whereas routine tests with single stimulation elicited no defects. Extinction, however, was not always present. Later in the convalescent period she frequently perceived both stimuli, but in her attempts to localize the sensation on the left she often made errors. When nonhomologous regions were simultaneously stimulated, she perceived the two stimuli but frequently mislocalized the one on the left side. The direction of the mislocalization was ipsilateral and toward the level at which the normal side was being stimulated. When the right side of the face and the hand on the left side of the body were simultaneously tested, displacement occurred in more than 70 to 80 per cent of the test trials. Displacement was less prevalent in stimulus combinations of the right side of the face and parts of the left side of the body, exclusive of the hand. In stimulus combinations of the right side of the body and the left side, exclusive of the face, displacement was also less prevalent. In other words, it was in the stimulus combination of the right side of the face and the left hand that displacement was most apparent. Such a stimulus combination shows the most conspicuous gradient in excitability.

Comment.—Although the sensation displaced was always the one evoked in a localized region on the affected side of the body, the direction of the displacement was either ipsilateral or contralateral. In this, as in many other cases, contralateral displacement was not as common as ipsilateral displacement. Contralateral displacement became most apparent when method A (simultaneous stimulation of bilateral homologous regions) or method C (simultaneous stimulation of ipsilateral regions) was used. Case 4 illustrates the various types of displacement which may be observed by using the three methods of simultaneous stimulation.

CASE 4.—H. S., a right-handed man aged 52, was admitted to Bellevue Hospital with a history of two attacks of left hemiparesis and one attack of right hemiparesis in the preceding three years. There was also progressive mental deterioration.

Examination disclosed signs of bilateral cerebral involvement. There were spastic weakness of the left upper extremity, a Babinski sign bilaterally and a right homonymous field defect. The patient showed many interesting sensory phenomena, the details of which have been published elsewhere.[3] Among the prominent sensory disturbances were extinction and displacement

3. Bender, M. B.; Shapiro, M. F., and Teuber, H. L.: Allesthesia and Disturbance of the Body Scheme, Tr. Am. Neurol. A. **73**:170, 1948; abstracted, J. Nerv. & Ment. Dis. **108**:253-259, 1948; Arch. Neurol. & Psychiat. **62**:222-231 (Aug.) 1949.

of sensation. Extinction was found either with stimulation of bilateral homologous areas or with simultaneous stimulations of unilateral areas. When the patient reported that he felt both stimuli, he would frequently mislocalize or displace the one in the more affected sensory region (methods B and C in the following tabulation).

Method A

Region Stimulated	Area of Perception
Right and left sides of face	One on each side of face
Right and left hands	Both on left hand
Right and left forearms	Both on left forearm
Right and left sides of trunk	Both on left side
Right and left knees	Right and left knee

Method B

Right side of head and left hand	Right and left side of head
Right side of face and left hand	Right and left side of face
Right hand and left side of face	Left hand and left side of face or both on left side of face
Right side of face (scratching) and left hand (pinprick)	Scratch on right and scratch on left side of face
Right side of chest and left ankle	One on left side of chest; other on left elbow
Right hand (rubbing) and left ankle (pinprick)	Both on left hand (rubbing and pinprick)
Right knee and left side of abdomen	Both on left side of abdomen

Method C

Right side of face (rubbing) and right hand (pinprick)	Rubbing on right and left sides of face
Right side of neck and right hand	Right and left sides of neck
Right hand (pinprick) and right thigh (scratch)	Two scratches, both on left hand
Right forearm (rubbing) and right ankle (pinprick)	Rubbing on left forearm

Combinations of stimuli on the left side of the body always revealed extinction, with definite and consistent face dominance. There was no displacement when the left side only was stimulated.

Displacement was found with single or with double simultaneous stimulation. With single stimulation of the right upper extremity, the displacement was always referred to the contralateral (left) side. Tactile or painful stimuli applied to regions of the right upper extremity were always mislocalized to a corresponding point on the left upper extremity. With double simultaneous stimulation the displacement of sensation on the right was either contralateral or ipsilateral, depending on the method employed. Displacement was noted only when the patient did not report extinction of the sensation.

With method A, or simultaneous stimulation of homologous parts of the body, the patient's response showed displacement of sensation across the midline. This crossed displacement occurred only when the upper extremities or both sides of the trunk were tested. There was no displacement in tests of the face or the lower extremities. During the course of the illness the cutaneous area in which crossed displacement could be obtained became smaller and was confined to the right upper limb. With single stimulation applied to the right upper extremity, he invariably indicated the sensation as being on the left. This is an example of true allesthesia, confined to tests of one region of the cutaneous sensory field. When the right and the left upper extremity were tested simultaneously, the patient indicated he felt both sensations on the left side. Stimulation of both arms, forearms or hands resulted in localization of both sensations to the corresponding region on the left side. There was no mislocalization when parts of the body other than the right upper extremity were tested.

With method B, or simultaneous stimulation of nonhomologous regions, there was ipsilateral displacement. On stimulation of the right side of the face and the left hand, the patient reported he felt a sensation on each side of the face. This is an example of ipsilateral displacement of sensation. This ipsilateral displacement to the left side of the face of the sensation evoked in the left hand seemed to have been influenced by the stimulation made on the right side of the face.

With method C, or simultaneous stimulation of parts on the same side of the body, the patient showed crossed or diagonal displacement. On stimulation of the right side of the face and the right hand by pinprick, the patient indicated that he felt the pricks in the right and left sides of the face. Light tapping on the right side of the face and pinprick applied to the right hand were perceived as tapping on each side of the face. The displacement in each instance was from the right hand across the midline and up to the left side of the face. Apparently, the stimulus in the right side of the face influenced even the quality of sensation evoked in the right hand; i. e., the patient perceived tapping on each side of the face rather than pinprick. The sensation evoked with pinprick in the right hand was displaced across the midline and up to the left side of the face. The tapping applied to the right side of the face seemed to have influenced the direction of the displaced sensation. The stimulus to the right side of the face was the most dominant, stable and well localized. Method C also showed face dominance or hand extinction, so that stimulation of the right side of the face and right hand resulted in a sensation in the face but not in the hand.

It was previously shown that the phenomenon of extinction became more manifest when the patient was under the influence of drugs. Alteration in the conditions of testing by the intravenous administration of amobarbital sodium caused a spread of the cutaneous area from which extinction could be obtained. My colleagues and I found that amobarbital sodium had a similar influence on the phenomenon of displacement. In this case, amobarbital sodium produced spread of the area which had demonstrated the phenomenon of displacement. A few minutes after intravenous injection of 0.4 Gm. of the drug simultaneous stimulation of homologous regions on the two sides of the face or trunk or on the upper limbs resulted in crossed displacement. The displacement was directed toward the left side of the body. Also, while under the influence of amobarbital, the patient showed a spread of the area from which extinction could be obtained on stimulation of parts on the left side of the body.

Just as in extinction, the occurrence of displacement was influenced by the timing of application of the stimulus. This is illustrated in studies of the following case.

CASE 5.—J. M., a man aged 43, was transferred to Bellevue Hospital with a history of alcoholism and of showing bizarre behavior for five months. The psychiatric diagnosis was schizophrenia.

Examination showed symptoms of severe mental changes and numerous disorders in sensation. The patient perceived all sensations elicited with the method of single stimulation. However, it was noted that every sensation evoked by a stimulus applied to the left upper extremity was mislocalized to the homologous region on the right side. This response is an instance of allesthesia, or crossed displacement, with the method of single stimulation. Simultaneous stimulation of the right and the left side of the body showed defects which always occurred on the left side of the body. Errors were not made in his responses to stimulations on the right side. With simultaneous stimulations the defects elicited were extinction and ipsilateral displacement on the left side of the body and contralateral displacement from left to right. It is not necessary to describe here the extinction phenomenon. Suffice it to say that this was found on repeated tests and showed all the characteristics previously described in other cases. The details of this case have been published in another communication.[4] When the patient felt both stimuli, the sensation on the left was mislocalized; that on the right side was always correctly placed. In tests with method B, or the simultaneous stimulation of the right side of the face and the left hand, there was ipsilateral displacement of sensation from the left hand to the left side of the face. In tests with method A, or simultaneous stimulation of the hands, the patient frequently indicated that he felt a sensation only on the right side; extinction was present on the left. Occasionally he felt two sensations, which he localized to the right. There was crossed displacement from left to right. Crossed displacement became much more apparent and occurred more frequently when the timing of the two stimulations was changed. Instead of the stimulations being simultaneous, they were made in quick succession. When this was done; the patient felt both sensations. Thus, on stimulation, first, of the left hand and, after an interval of one second, of the right hand, he reported

4. Bender, M. B., and Nathanson, M.: Patterns in Allesthesia and Their Relation to Disorder of Body Scheme and Other Sensory Phenomena, Arch. Neurol. & Psychiat. **64**:501-515 (Oct.) 1950.

that he felt two sensations in the right hand. Evidently the sensation on the left side was displaced to the right. However, when the order of stimulation was first the right and then the left hand, his responses and localization of sensation were correct. In short, this patient showed extinction, ipsilateral displacement and contralateral displacement whenever the left upper extremity was compared with any region on the right side of the body. Each of the responses depended partly on the method of testing employed. The responses were consistently obtainable only when the left upper extremity was one of the two areas tested. These changes were more apparent in the hand and less evident in the arm and shoulder.

Comment.—Although the brain was bilaterally involved, the various methods of testing demonstrated a difference between the two sides of the body. The sensory reactions on the right dominated over those on the left. Sensations originating from the left side of the body were either imperceptible or, if perceived, poorly localized. The sensations on the left were under the constant influence of those originating from the right, or normal, side. The displacement of sensation, whether ipsilateral or contralateral, showed a predictable direction and seemed to be determined by stimuli originating from the right side of the body.

3. Displacement of Sensation in Patients with Lesions of the Spinal Cord.—As in the case of extinction, the phenomenon of displacement is not limited to patients with lesions of the cerebral hemispheres. We have noted the occurrence of displacement in patients with lesions in the spinal cord who showed sensory disturbances but no evidence of disease of the brain. The following case illustrates this point.

CASE 6.—A coal miner aged 43 was admitted to the New York University Hospital with a history of progressive numbness and weakness in the left lower extremity for eight months. Owing to an injury at the age of 29, he had had a midthigh amputation on the right side. Examination disclosed paralysis of the left lower extremity and of the stump of the right thigh. Sensation for pain, temperature and vibration was bilaterally decreased below the level of the fourth thoracic segment of the spinal cord. Vibration and position sensibility was lost at and below the iliac crests. Mentally the patient was entirely normal.

A lumbar puncture and myelogram showed complete block at the fourth thoracic vertebra. A laminectomy was performed, and an extradural neurofibroma was removed. The patient recovered and was discharged from the hospital.

Prior to the operation the significant sensory changes to be described were noted. There was no question as to the level of the lesion elicited with the customary method of single stimulation. Noteworthy were the alterations found on double simultaneous stimulation of areas above and below the sensory level; of these, extinction was foremost. A single intense painful stimulus, such as a pinch or firm pinprick, was perceived below the level of the lesion. Vigorous or noxious single stimuli applied to the thigh or leg also evoked sensations. Nevertheless, as soon as any additional contact with the skin was made simultaneously or successively above the level, he reported that he felt only one sensation and localized it above the level of the lesion. Extinction occurred even while the patient watched the application of the stimuli. In fact, he was amused that a sensation which he perceived with single stimulation below the level of the lesion "disappeared" as soon as another sensation was evoked on stimulating any point above the level. The sensation which was retained, i. e., "dominant" one, was always correctly localized.

Extinction was not a consistent finding. At times the patient felt both stimuli. This was especially apparent when a vibrating tuning fork was applied below the level of the lesion and another stimulus (pinprick) above the level. When this vibrating stimulus was simultaneously compared with another stimulus applied above the level of the lesion, he displaced the vibration sense to a point at or near the locus of sensation evoked above the level of the lesion. Thus, a vibration stimulus at the ninth rib and a pinprick stimulus at the clavicle were perceived as a tingling sensation (or "stick and buzz," as the patient said) at the clavicle. A tactile stimulus at the right clavicle and application of a vibrating tuning fork at the lower rib on the left side were reported, to quote the patient, as "buzz and a touch" at the right clavicle. It was not a

matter of stimulations being made within an area of a few spinal segments, for simultaneous stimulation of regions far above and below the level yielded similar results. Thus, a pinprick stimulus on the left cheek and a vibration stimulus at the seventh rib on the right side were reported as a "stick and a buzz" on the left cheek. Pinprick on the right shoulder and a vibration stimulus at the sixth rib on the left side were reported as "stick and buzz" on the right arm, and so on. It should be noted that in all of these tests displacement, when it occurred, was always toward the location of the sensation felt by the patient above the level of the lesion, whether it was homolateral or contralateral to the stimulus applied below the level of the lesion. Displacement was present with the patient's eyes closed or open and even while he looked at the point of stimulation.

Comment.—In order to evoke displacement in cases of spinal cord disease, the stimuli must be applied simultaneously above and below the level of the spinal lesion. The sensation displaced is the one evoked in the area with less sensation, i. e., the defective area, and this displacement is directed toward the part of the body stimulated above the level of the lesion, or the normal sensory region.

This patient also showed several interesting sensory phenomena below the level of the lesion. Thus, on simultaneous stimulations in areas below the level, there were (*a*) extinction across the midline of the body; (*b*) extinction along the long, or vertical, axis with rostral dominance, i. e., with the trunk dominating over the lower extremity when both were simultaneously stimulated; (*c*) poor localization of sensation with displacement which showed no consistency in direction (a pinch of skin in the left flank was localized as a touch sensation on the right side of the abdomen [5]); (*d*) pronounced fluctuation and instability of threshold of sensation, and (*e*) illusory movements (a stationary noxious stimulus on the thigh was described as a sensation moving along the abdomen).

Phenomenon of Displacement Brought Out with Face-Hand Test in Patients Recovering from Acute Anesthesia and Electroshock Stimulation: Besides the relatively lowered threshold and spatial dominance, as illustrated in the cases of lesions of the cerebral and spinal cord, there were other factors which influenced the occurrence of displacement. As previously stated, intensity and duration of stimulations, timing of stimulations and effects of drugs are but a few examples. In fact, all situations which tended to produce the phenomenon of extinction also influenced the occurrence of displacement. Recently we have studied subjects who were recovering from a general anesthetic. These patients showed a high incidence of extinction or of displacement when the face and one hand were simultaneously tested. The phenomena were apparent in the first two or three hours of recovery from the anesthetic. Mentally, these subjects were confused and disoriented. However, in spite of their mental state, the subjects showed order in their reactions to simultaneous stimulations. An analysis of all reactions revealed the same pattern as that found in the normal adults and children and in patients with an organic mental syndrome. The incidence of the phenomena of extinction and displacement

5. This form of displacement has previously been observed in the experimental monkey (Dusser de Barenne, J. G.: Zur Kenntnis der Alloasthesie. Experimente und Betrachtungen, Monatsschr. Psychiat. u. Neurol. **34**:523-540, 1913. Mott, F. W.: Results of Hemisection of the Spinal Cord in Monkeys, Phil. Tr. Roy. Soc. **183**:1-59, 1892) and in man (Holbrook, T. J., and de Guiterrez-Mahoney, C. G.: Diffusion of Painful Stimuli over Segmental, Infrasegmental and Suprasegmental Levels of the Spinal Cord, abstracted, Federation Proc. **6**:131, 1947; Personal communication to the author. Ray, B. S., and Wolff, H. G.: Studies on Pain: "Spread of Pain"; Evidence of One Site of Spread Within the Neuraxis of Effects of Painful Stimulation, Arch. Neurol. & Psychiat. **53**:257-261 [April] 1945).

was high in subjects who were recovering from a general anesthetic. It must be emphasized here that these very subjects showed normal mental function and responses with double simultaneous stimulation of the face and hand before and after full recovery.

In several instances patients who had just recovered from a convulsion produced by electric stimulation of the brain were studied with double simultaneous stimulation. These patients also showed reactions which were similar to those of patients with an organic mental syndrome. They manifested a high incidence of extinction and displacement, just as did the subjects recovering from anesthesia. When an anesthetic, such as amobarbital sodium or thiopental sodium, 0.5 Gm., was administered intravenously just prior to the electrically induced convulsion, the incidence of extinction or displacement in the hand was almost 100 per cent. This phenomenon became more conspicuous with each successive convulsion, particularly when an organic mental syndrome became very apparent.

4. *Phenomenon of Displacement in Normal Subjects.*—Adults: As already mentioned, displacement was found in the normal adult and child.[2c] In the adult the incidence was low. Displacement occurred on the first trial and was often no longer apparent on the second trial. The mislocalized stimulus or the displaced sensation was usually perceived as vague and indefinite, and the response showed a long latency. Once the normal adult learned that he was being stimulated in two places and that he was expected to localize the points of stimulation, he seldom made errors after the first two trials. There were some subjects who were apparently normal but who became tense and apprehensive when confronted with the examinations. These subjects tended to make more errors on double simultaneous stimulation with face-hand test. The errors were either imperception in the hand or, less frequently, displacement from hand to face. At times these subjects had as many as six to 10 trials before they learned the proper "set." Once the "set" was learned, they made no errors.

Children: In children, particularly the group between the ages of 3 and 6 years, the incidence of displacement was much higher. Moreover, the phenomenon was present on repeated occasions and on examinations performed on different or subsequent days. These young children were told that they were to be touched in two places, and even then one of the sensations, usually the one evoked in the hand, was displaced to the face. This was apparent even on stimulation of the same side. Thus, when we touched the left side of the face and the left hand of a 3 year old child, the little girl replied that she felt one stimulus on the left cheek and indicated the other as being on the left side of the chin. Subsequent examinations with face-hand combinations, crossed or uncrossed, showed that she perceived only on the face in almost every test. On the following day similar tests again showed displacement when the right side of the face and the left hand were tested. The child now pointed to the right side of the face, and when asked where else she felt the stimulus she pointed to the left side of the face.

Children above the age of 6 years showed a lower incidence of displacement and extinction. The more mature and the older the child the greater the tendency to yield responses which were closer in type and frequency to those found in the adult. In fact, preliminary statistics show that the lowest incidence, less than 40 per cent of extinction or displacement, is found on initial face-hand tests at puberty and during the adolescent years.

GENERAL COMMENT

Displacement of sensation is a disorder in localization. The characteristic feature is that the mislocalization is usually in a predictable direction and is most apparent on double simultaneous stimulation. The direction of the displacement is determined by the configuration of two simultaneously applied stimuli. Thus, when the right side of the face and the left hand are touched, it can be predicted that the sensation evoked in the left hand will be displaced upward to the left side of the face. From the foregoing observations, it may be concluded that the displacement to the left side of the face is influenced by the stimulus applied to the right side of the face.

Studies of various combinations of double simultaneous stimulations of different areas of the body disclosed patterns in which some parts of the body dominate over others. In summary, it was found that the face is most, and the hand is least, dominant. The order of dominance for other parts of the body lies between the face and the hand. Tests with combinations of stimulations of parts of the body other than the face and hand showed the incidence of displacement to be variable. Here, too, the stimulus in the dominant area usually determines the direction of the displacement.

Displacement of sensation is not a mere pathological curiosity. In can be found in the normal adult, and particularly in young children. Moreover, the patterns of dominance and displacement observed in the normal subject are identical with those which occur in the patient. It is significant that the incidence of displacement in the normal young child is almost the same as that found in patients with diffuse disease of the brain.

A type of displacement of sensation which has been known for many years is allesthesia, or displacement across the midline of the body.[6] Allesthesia can be obtained with single stimulation. Thus, when a single stimulus is applied to one region of the body, it is sometimes localized to the corresponding region on the opposite side of the body. The displacement is usually away from an area which is deficient in sensibility toward an area on the opposite side of the body which is relatively normal.

Instances of allesthesia obtained with single stimulation have been described on numerous occasions. However, there have been few, if any, reports on the types of displacement described in this communication, namely, those obtained on double simultaneous stimulation with ipsilateral displacement or displacement from the hand into the extrapersonal space. Specifically, the subject is unable to localize the stimulus on the hand. Instead, he mislocalizes the stimulus to the region on which the hand rests, i. e., another part of his body, such as the thigh, or a table or even into space. Jones[7] mentioned a type of mislocalization occurring on stimulation of the hand which he called dyschiria. This term is not altogether adequate, but if it is used to mean that there is displacement of sensation away from the hand, it should be understood as such.

Thus far, cases of displacement on double simultaneous stimulation have been found in tests of cutaneous sensation. As yet no concerted attempt has been made to investigate cases of displacement in the visual or auditory modalities by using

6. Obersteiner, H.: Über einige Sensibilitätsstörungen bei Neurosen, Wien. med. Presse **21**:1635, 1880; On Allochiria: A Peculiar Sensory Disorder, Brain **4**:153-163, 1881-1882.

7. Jones, E.: The Pathology of Dyschiria, Rev. Neurol. & Psychiat. **7**:499 and 599, 1909.

simultaneous stimulation methods. Preliminary tests, however, have shown that ipsilateral displacement can occur in the vertical plane in tests of one half-field of vision. There are no data on ipsilateral displacement in hearing. Instances of crossed displacement on double simultaneous stimulation tests in vision, as well as in hearing, have been described. Contralateral displacement in vision with tests of single stimulation has been known since 1888 as optic or visual allesthesia.[8] Similar observations have been made in tests of hearing since 1888.[9] The phenomenon of displacement should not be confused with disorder in topical localization, or spot finding, as described by Head.[10] The mislocalization which occurs in defective spot finding is haphazard and does not show a predictable direction such as that noted in the phenomenon of displacement.

The significance of the phenomenon of displacement is the same as that for the phenomenon of extinction.[11] Each phenomenon implies that when a combination of stimuli are applied simultaneously one of the sensations evoked influences the other; i. e., the sensation evoked by one stimulus may be extinguished, obscured or displaced by a simultaneous stimulus, depending on which parts of the sensory field are tested. This influence shows a distinct spatial pattern. These patterns are consistent and resemble those noted in other altered states, such as in hemiplegia. It is significant that the same patterns are found in the normal subject. On correlating these facts, one may imply that the patterns seen in disease are the same as those found in the normal state. Study of pathological states, therefore, should give one insight into normal function.

Another implication from the foregoing observations is that any one sensation which may be evoked is altered to some extent by the constant stream of stimuli which are present in the background, stimuli which are continuously arriving from within and without the organism. Such hypotheses have been proposed by Lashley,[12] Goldstein[13] and Klüver.[14] When a sensory area is stimulated, the impulses enter into a nervous system which is in a constant state of organized activity. The locus of a sensation is thus determined by a background which, although not static, is stable and patterned in its inner relationships. One pattern which is found in the organization of sensation is that which was described in this communication, namely, face dominance with either extinction or displacement in the hand when the face and hand are simultaneously stimulated.

Explanations or reasons for such organization and patterns are beyond the scope of this paper. There is no one current theory which would satisfactorily

8. Magnin, P.: Allochirie visuelle chez une hystérique hypnotisée, Thesis, Paris, 1889; Compt. rend. Soc. biol. **5**:57, 1888. Hermann, G., and Pötzl, O.: Die optische Allöasthesie: Studien zur Psychopathologie der Raumbildung, Berlin, S. Karger, 1928.

9. Gellé: Un cas d'allochirie auditive, Gaz. d. hôp. **61**:92, 1888.

10. Head, H.: Studies in Neurology, London, Oxford University Press, 1920, vol. 2.

11. Wortis, S. B.; Bender, M. B., and Teuber, H. L.: The Significance of the Phenomenon of Extinction, J. Nerv. & Ment. Dis. **107**:382-387, 1948.

12. Lashley, K. S.: Integrative Functions of the Cerebral Cortex, Physiol. Rev. **13**:1-42, 1933; The Problem of Cerebral Organization in Vision, Biol. Symposia **7**:117-164, 1942; Functional Interpretation of Anatomic Patterns, in Patterns of Organization in the Central Nervous System, A. Res. Nerv. & Ment. Dis., Proc., 1950, to be published.

13. Goldstein, K.: The Organism: A Holistic Approach to Biology Derived from Pathologic Data in Man, New York, American Book Company, 1939.

14. Klüver, H.: Behavior Mechanisms in Monkeys, Chicago, University of Chicago Press, 1933, pp. 17 and 387.

explain a pattern in which the face is most dominant and, at the same time, the hand is least dominant. Perhaps accumulation of additional data may make it possible to formulate a working hypothesis for patterns in organization of sensory function.

SUMMARY AND CONCLUSIONS

Observations on patients with the method of simultaneous stimulation of the face and hand disclosed two types of abnormal responses: (*a*) imperception of the hand stimulus and (*b*) mislocalization of the hand stimulus.

The mislocalization showed direction and predictability. For this reason this effect has been called the phenomenon of displacement.

The displacement occurred (*a*) in an ipsilateral direction, this being the most frequent type; (*b*) in a contralateral direction, or (*c*) outward into the extrapersonal space, this being the least frequent type.

The direction of the ipsilateral displacement was usually toward the "dominant" sensory region. The direction of contralateral displacement was away from the "bad," or hypesthetic, side of the contralateral (normal) regions. In general the direction of the displacement was determined by the degree of dominance of the sensory areas tested. The order of dominance was established after numerous combinations of simultaneous stimulation of two different regions of the body. The face was most dominant, whereas the hand was least dominant.

The phenomenon of displacement was found not only in patients with disease of the brain but also in patients with disease of the spinal cord. Most significant is the observation that the phenomenon of displacement occurs in a small percentage of normal adults, in a high percentage of normal adults during the period of recovery from anesthesia and in a high percentage of normal young children.

The significance of the phenomenon of displacement is the same as that of extinction. The occurrence of the phenomenon of displacement or extinction suggests that a sensation evoked by one stimulus is constantly influenced by other stimuli. Perception is organized in characteristic patterns. The pattern found in patients with disease of the nervous system is the same as that which one may observe in the normal subject.

REFLEXES EVOKED BY COLD STIMULI IN INJURIES OF THE SPINAL CORD

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INTRIGUED by the challenge of the article by Macht and Kuhn,[1] in which it is pointed out that there is a paucity of information on the reflexes evoked by cold stimuli in spinal man, we studied such responses in our material of injuries to the spinal cord.

MATERIAL AND METHODS

One hundred and fifty-two patients were studied, 43 with lesions of the cervical portion of the spinal cord, 69 with lesions of the thoracic segments and 40 with lesions of the lumbar segments. At times, for some particular investigation, such as that of reflex activity in the upper extremities from stimulation of the upper extremities, a smaller number of patients were studied with special attention to this detail, and the number will be indicated in appropriate paragraphs.

The stimulus consisted of the application of a wisp of cotton saturated with ether. The inefficacy of touch itself was determined by stimulation with a dry wisp of cotton applied in a similar manner to similar areas.

Several areas were stimulated to determine the most effective zone, or the area which was productive of the greatest number of reflex movements. In the case of lesions of the thoracic and lumbar portions of the spinal cord, the areas stimulated included the sole, the inner and outer surfaces of the leg and thigh, the anterior surface of the thigh and the abdominal wall. In the case of lesions of the cervical portion of the spinal cord, the areas stimulated included the volar and dorsal surfaces of the arm, forearm and hand, in addition to the areas already mentioned.

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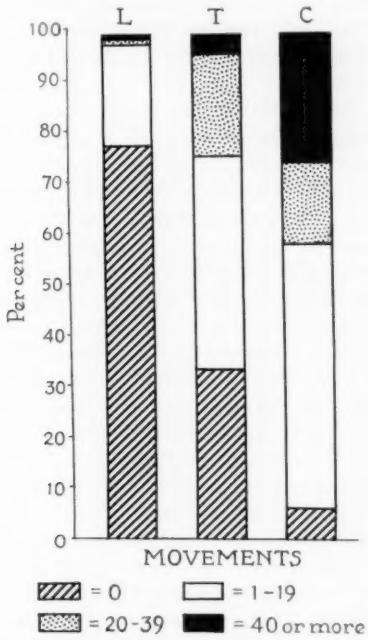
1. Macht, M. B., and Kuhn, R. A.: Responses to Thermal Stimuli Mediated Through the Isolated Spinal Cord, Arch. Neurol. & Psychiat. **59**:754 (June) 1948.

TOTAL ACTIVITY OF THE INJURED SPINAL CORD

Fifteen movements of segments about joints of a lower extremity may result from stimulation of suitable areas. These consist of flexion of the toes, foot, leg and thigh; extension of the toes, foot, leg and thigh; inversion and eversion of the foot; abduction and external rotation of the thigh; adduction and internal rotation of the thigh, and a Babinski-like response. These movements may occur as flexor or extensor responses or may be multiple when successive or crossed reflexes are present. The total number of movements ensuing as the result of stimulation of the seven areas mentioned constituted an index of total activity of that portion of the injured



Percentage of numbers of movements evoked by stimulation with cold in cases of spinal cord injuries at various levels.

spinal cord which was distal to the site of injury. This index of activity was then assessed with the level of the injury.

Failure to respond by movement to cold stimuli was noted in 6 per cent of cervical lesions, 33 per cent of thoracic lesions and 77 per cent of lumbar lesions of the cord. A total response of one to 19 movements was present in 52 per cent of cervical lesions, 42 per cent of thoracic lesions and 20 per cent of lumbar lesions of the cord. A total response of 20 to 39 movements was present in 16 per cent of cervical lesions, 20 per cent of thoracic lesions and 1 per cent of lumbar lesions. A total of 40 or more movements was noted in 25 per cent of cervical lesions, 4 per cent of thoracic lesions and 1 per cent of lumbar lesions (chart). From

this it is seen that the total activity of the spinal cord is greater in cervical than in thoracic injuries of the spinal cord and least in the lumbar injuries. This corresponds to a former study [2] using other types of stimuli, such as scratch.

THE REFLEXOGENOUS ZONES

The reflexogenous zones for cold stimuli in cases of injury of the cervical portion of the spinal cord were effective in the following order: sole, inner surface of the thigh, inner surface of the leg, outer surface of the leg, anterior surface of the thigh and outer surface of the thigh. For thoracic lesions, the order was as follows: sole, inner surface of the leg, inner surface of the thigh, outer surface of the leg, outer surface of the thigh and anterior surface of the thigh. For lumbar lesions, the order was as follows: sole, outer surface of the leg, inner surface of the leg, outer surface of the thigh, inner surface of the thigh and anterior surface of the thigh.

PATTERN OF RESPONSE

Crossed reflexes were induced in 13 instances, representing 30 per cent of 43 cases of lesions of the cervical portion of the spinal cord; in 10 instances, representing 14.6 per cent of 69 cases of lesions of the thoracic portion, and in 3 instances, representing 7.5 per cent of 40 cases of lesions of the lumbar portion.

The relative frequency of muscle group contractions in ipsilateral reflexes was observed in the following descending order: extensors of the toes, flexors of the thigh, flexors of the leg, dorsal flexors of the foot, a Babinski-like response, flexors of the toes, adductors of the thigh, invertors of the foot, extensors of the thigh, plantar flexors of the foot, internal rotators of the foot, extensors of the thigh, external rotators of the thigh, abductors of the thigh and evertors of the foot.

Dorsal flexion of the toes, dorsal flexion of the feet and flexion of the leg and thigh occurred in 1,056 instances, as compared with 197 cases of plantar flexion of the toes, plantar flexion of the foot and extension of the leg and thigh.

Many seemingly unrelated contractions of muscle groups occurred, as in flexors of the toes, invertors of the foot and extensors of the leg; or in flexors of the toes, invertors of the foot and flexors of the thigh; or in extensors of the leg and thigh and adductors of the thigh; or in dorsal flexors of the foot, and flexors of the leg and thigh; or in extensors of the toes, dorsal flexors of the foot and flexors of the leg and thigh; or in flexors of the toes, foot, leg and thigh; or in extensors of the leg and external rotators of the thigh; or in flexors of the toes, extensors of the leg and extensors and external rotators of the thigh. The character of the response was not related to the completeness or incompleteness of the lesion.

LESIONS OF THE CERVICAL PORTION OF THE SPINAL CORD

A special study was made of 14 cases of lesions of the cervical portion of the spinal cord with reference to reflexes in the upper extremities evoked by stimulation of the skin of the arm, forearm and hand. The arm and forearm were found to be more effective reflexogenous areas than the hand. Reflex movements evoked by stimulation of either the arm or the forearm were obtained in 13 cases and by stimulation of the hand in only seven cases.

2. Pollock, L. J.; Boshes, B.; Arieff, A. J.; Newman, L. B.; Chor, H.; Brown, M.; Finkelman, I., and Kostrubala, J. G.: Injuries to Spinal Cord, Tr. Am. Neurol. A. **74**:216, 1949.

Stimulation of the volar surface of the arm, forearm and hand evoked more reflex movements than stimulation of their dorsal surface. Thus, in the arm 12 responses were evoked from volar and 5 from dorsal stimulation; in the forearm, 11 from volar and 8 from dorsal stimulation, and in the hand 7 from volar and none from dorsal stimulation.

The responses included contraction of several muscle groups, a total of 58 movements being recorded in a group of 14 cases analyzed. Of the 17 movements about the shoulder, 10 were evoked from stimulation of the arm, 6 were evoked from the forearm and 1 was evoked from the hand. Of the 2 movements about the elbow, 1 was evoked from the arm and 1 from the forearm. Of the 10 movements about the wrist, 5 were evoked from the arm, 4 were evoked from the forearm and 1 was evoked from the hand. Of the 6 movements about the thumb, 2 were evoked from the arm, one was evoked from the forearm and 3 were evoked from the hand. Of the 23 movements about the fingers, 7 were evoked from the arm, 10 from the forearm and 6 from the hand.

Of the 25 movements of the upper extremity obtained from stimulation of the arm, 10, representing 40 per cent of the movements, were about the shoulder; 1, representing 4 per cent, was about the elbow; 5, representing 20 per cent, were about the wrist; 8, representing 8 per cent, were about the thumb, and 7, representing 28 per cent, were of the fingers.

Of the 22 movements of the upper extremity obtained from stimulation of the forearm, 6, representing 27 per cent of the movements, were about the shoulder; 1, representing 5 per cent, was about the elbow; 4, representing 18 per cent, were about the wrist; 1, representing 4.5 per cent, was about the wrist, and 10, representing 45 per cent, were of the fingers.

Of the 11 movements of the upper extremity obtained from stimulation of the hand, 1, representing 9 per cent of the movements, was about the shoulder; none were obtained about the elbow; 1, representing 5 per cent, was about the wrist; 3, representing 27 per cent, were about the thumb, and 6, representing 54 per cent, were of the fingers.

The reflexogenous zone seemed to be less segmental than proximal and distal. At times movements of the thumb and fingers were elicited from the hand alone, the forearm alone, the arm alone or from either the arm or the forearm.

The order of frequency of movements was adduction of the shoulder, flexion of all or some of the fingers, extension of all or some of the fingers, adduction of the thumb, radial abduction of the wrist, ulnar deviation of the wrist, depression of the shoulder, flexion of the wrist and flexion of the thumb. Adduction of the arm was associated with both flexion and extension of the fingers.

In one case, a crossed reflex ensued from stimulation of the forearm, producing ipsilateral flexion and contralateral extension of the fingers. In the same case, with stimulation on the other side, ipsilateral extension and contralateral flexion of the fingers occurred.

In this special study of 14 cases of cervical lesions of the cord, responses evoked by stimulation of reflexogenous zones below the motor innervation were noted. In 3 of these cases ipsilateral responses resulted from stimulation of the skin of the inner surface of the thigh, the upper part of the abdomen and the costal margin; in 2 of these 3 cases movement of the shoulder, and in 1 case flexion of the thumb and

fingers, occurred. In 3 cases, including 1 of these 3 cases, a crossed reflex occurred from stimulation of the inner surface of the thigh, the abdomen and the costal margin. In 1 case, adduction of the arm, and in others extension of the wrist, fingers and thumb, occurred.

Since movement of the fingers is produced by stimulation of the outer surface of the arm or of the abdomen, in instances in which these areas are respectively above or below the level of the lesion of the spinal cord, it would appear that with partial lesions radiation occurs upward as well as downward in the spinal cord.

UPPER LEVEL OF REFLEXOGENOUS ZONES PRODUCING REFLEXES IN THE LOWER EXTREMITIES

Reflex activity in the lower extremities was evoked by stimulation of the arm in one case of injury to the cervical portion of the cord, and of the arm, forearm and hand in another case. In both cases these reflexes were either ipsilateral or became crossed in the course of successive reflexes.

Reflex activity in the lower extremities evoked by stimulation of the abdominal wall was present in 18 of 43 cases of injury to the cervical portion of the cord. A total of 25 responses was obtained from stimulation of the upper abdominal quadrants, and 11 responses, from stimulation of the lower quadrants.

Crossed responses developing in the course of successive reflexes occurred in 14 cases. When ipsilateral flexion occurred, the crossed reflex in the majority of cases was also flexor in type. Of all ipsilateral and crossed reflexes, only seven were of the extensor type.

Although a crossed gluteal reflex from stimulation of the buttock with cold [3] has been noted previously in a few cases irrespective of the level of the lesion, no such observation was made in the present study, and stimulation of the buttocks, when effective, produced a contraction of the ipsilateral gastrocnemius.

SUMMARY

As was expected, the stimulus of cold, as in the case of other exteroceptive stimuli, produced reflex activity from the distal end of an injured spinal cord.

The effectiveness of the stimulus was less than that of scratch or of multiple pinprick. The responses occurred from stimulation of similar reflexogenous zones.

As was the case with other stimuli, the greatest number of muscle groups participating in the reflexes occurred with injury of the cervical portion of the spinal cord; next, with injury of the thoracic portion, and least, with injury of the lumbar portion. The greatest number of cases in which no response followed stimulation was that of injuries to the lumbar portion of the spinal cord; next, that of injuries to the thoracic portion, and least, that of injuries of the cervical portion. The frequency of crossed reflexes was least with lesions of the lumbar portion of the spinal cord, next with lesions of the thoracic region and greatest with lesions of the cervical region. Ipsilateral reflexes alone predominated.

With lesions of the cervical portion of the spinal cord, abnormal reflex activity in the upper extremities occurred from stimulation of the skin of the upper extremities. The arm and forearm were more effective reflexogenous zones than the hand, and the volar surfaces were more effective than the dorsal surfaces. The arm and

3. Lawn, H. J.: Crossed Gluteal Reflexes, Arch. Neurol. & Psychiat. **63**:334 (Feb.) 1950.

forearm were the most effective zones for reflex movements of the shoulder, forearm and wrist; and the hand was the most effective for movements of the thumb and fingers. Ipsilateral and contralateral reflexes of the upper extremities were evoked from stimuli below the level of the lesion, in a few cases as low as the inner aspect of the thigh. Stimulation of the upper extremity resulted in reflex movements of the lower extremities in two instances, representing 5 per cent of 43 cases, whereas stimulation of the abdominal wall resulted in such reflex activity in 18 instances, representing 41 per cent of 43 cases of lesions of the cervical portion of the cord. More reflex activity resulted from stimulation of the upper than of the lower abdominal quadrants.

SUBARACHNOID HEMORRHAGE IN MELANOMA OF THE BRAIN

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GARIN, Plauchu and Masson [1] reported four attacks of subarachnoid hemorrhage in a case of melanocarcinoma of the jaw with multiple metastatic melanomas in the meninges and brain, observed at necropsy. Other investigators have since commented on the occurrence of bloody or xanthochromic cerebrospinal fluid in cases both of primary and of secondary melanoma of the brain. Schnitker and Ayer [2] found grossly bloody fluid in three cases, xanthochromic fluid in two cases and clear fluid in seven cases of a series of primary melanomas of the nervous system. Loewenberg [3] stated that in 14 cases of metastatic melanoma of the brain collected from the literature, including six of his own, the cerebrospinal fluid was distinctly xanthochromic in seven and slightly yellowish in one; in several instances it was bloody. Wortis and Wortis [4] advised:

> Whenever bloody or xanthochromic fluid is found in a patient who has melanomatous tumors of the skin, the spinal fluid should be examined for the presence of melanin pigment or melanoma cells.

We report six cases of melanoma of the brain, with subarachnoid bleeding in five; four cases were verified histologically. A review of the literature to determine the incidence of subarachnoid bleeding in metastatic melanomas of the brain and a comparison with the frequency of subarachnoid bleeding in other metastatic brain tumors follow.

REPORT OF CASES

CASE 1.—L. S., a woman aged 55, was admitted to the Montefiore Hospital on Jan. 12, 1948, with a history of sudden weakness of the right side two months prior to admission and impairment of speech for a few minutes on two occasions, six and four weeks before admission. Many small nodules, the largest in the right groin, were noted over a period of 10 months. She had lost 25 pounds (11.3 Kg.) in one year. The patient was ambulatory when admitted to the hospital.

The examination on admission showed numerous nontender and nonpigmented subcutaneous nodules on the trunk and limbs and, on the right side, spastic hemiparesis, a more active knee jerk, poor plantar flexion and an organic hemisensory syndrome. An electroencephalogram

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From the Neuropsychiatric Services of the Montefiore Hospital for Chronic Diseases and the Morrisania City Hospital.

1. Garin, C.; Plauchu, M., and Masson: Naevo-carcinome de la joue avec métastases cérébrales se traduisant par un tableau d'hémorragie méningée à allure paroxystique, Lyon méd. **149**:803, 1932.

2. Schnitker, M. T., and Ayer, D.: The Primary Melanomas of the Leptomeninges: Clinico-Pathologic Study with Review of Literature and Report of an Additional Case, J. Nerv. & Ment. Dis. **87**:45, 1938.

3. Loewenberg, P. C.: Die Gehirnmetastasen der Melanome, Thesis, Berlin, 1939.

4. Wortis, H., and Wortis, S. B.: Metastatic Melanoma Involving the Central Nervous System, Arch. Neurol. & Psychiat. **36**:601 (Sept.) 1936.

showed abnormalities bilaterally. A lumbar puncture on January 14 showed clear fluid with an initial pressure of 104 mm. of water. Biopsy of one of the nodules in the left arm was reported as showing an amelanotic melanoma.

The weakness on the right side improved slightly, and the patient was discharged on January 23. Between January 22 and February 4, x-ray treatments were given to the left side of the head. On February 6 the patient complained of sudden severe pain in the left side of the head, followed by paralysis of the right extremities; she vomited soon afterward and became semistuporous. On her readmission, stupor and Cheyne-Stokes breathing were noted; the blood pressure was 90/50; the left pupil was wider than the right, and there was a Babinski sign bilaterally. A lumbar puncture on February 6 showed bloody fluid with 175,000 red blood cells and 26 white cells per cubic millimeter; the initial pressure was 440 mm. of water. The patient died six days later.

Autopsy of the brain, by Dr. M. G. Netsky, disclosed a small amount of blood in the subarachnoid space, especially over the right hemisphere. Six blackish nodules, varying in size from 0.2 to 1.5 cm. were observed. Three were in the left frontal lobe, two in the left posterior parietal region and one in the left temporal lobe. There were numerous fresh hemorrhages in the interior of the nodules. The report on microscopic sections by Dr. Netsky showed that the "cells were mostly large oval and vesicular or spindle-shaped. They were arranged in spoke-like fashion around blood vessels. The peripheral edge of the spokes was lined by mesothelium. An occasional cell contained brown pigment within the cytoplasm. Mitoses were numerous. A Gomori iron stain revealed a great deal of extranuclear iron, but the intracytoplasmic pigment was not stained. This indicated that the pigment was probably melanin."

CASE 2.—A woman aged 82 was admitted to the Morrisania City Hospital on Jan. 13, 1950, with a history of progressive weakness of the right extremities of six weeks' duration. Three years previously the patient noted a pea-sized, bluish, bleeding lesion of the left sole. She consulted dermatologists as the lesion continued to grow, and a diagnosis of malignant melanoma was made without biopsy. In view of the patient's age, no treatment was advised. The area of bluish discoloration enlarged to the size of a dime during 1947 and then remained stationary.

On admission the patient showed hemiparesis and an organic hemisensory syndrome on the right side and right homonymous hemianopsia with weakness of the left side of the palate and mild hoarseness. The blood pressure was normal.

Blood counts and the blood urea and sugar were normal. Roentgenograms of the skull and chest were normal. A spinal puncture revealed grossly bloody fluid with an initial pressure of 280 mm. of water and a total protein content of 140 mg. per 100 cc. A second puncture, performed four weeks later, showed a highly xanthochromic fluid. Melanin was not demonstrated in the urine or in the spinal fluid. Serologic reactions of the blood and spinal fluid were negative. Biopsy of the lesion in the left sole was reported at first as showing a "heavily pigmented nevus with no signs of malignancy."

The course of the patient's illness was downhill, and she died on March 20, 1950. Autopsy, by Dr. W. Aronson, showed a whitish nodule the size of a quarter, surrounded by dark hemorrhagic tissue, on the medial aspect of the left occipital lobe. Complete postmortem study revealed no other nodules. Histologic section of the brain nodule was reported as revealing an "amelanotic melanoma" by Dr. Maurice Richter and Dr. Maxwell Fein, of University Hospital, New York University.

CASE 3.—B. S., a man aged 36, was first admitted to the neurosurgical service of Dr. L. M. Davidoff at the Montefiore Hospital on Nov. 14, 1947, with a history of headaches and two convulsive seizures during the preceding eight months. Examination showed bilateral papilledema, left homonymous hemianopsia, bilateral nerve deafness and very mild weakness of the left side. A roentgenogram of the skull disclosed changes due to increased intracranial pressure. The electroencephalogram revealed a right temporal localization, and angiography showed upward displacement of the Sylvian vessels. A large hairy nevus was noted on the inner aspect of the distal half of one of the thighs; it was smooth, soft and only slightly raised above the skin. On November 20 a right lateral craniotomy was done, with complete removal of a large temporal tumor. Histologic examination, by Dr. M. G. Netsky, showed it to be a melanoma. The patient was given high voltage x-ray therapy and was discharged on December 1. He was readmitted on December 25, because of the recurrence of headache, nausea, vomiting and a

convulsive episode involving the left side, with olfactory hallucinations. Examination disclosed no bulging of the decompression and no papilledema. The left homonymous hemianopsia was still present, and the electrocortical changes were similar to those present during the first admission. Diphenylhydantoin was prescribed and x-ray therapy continued.

The patient did well for a short time after his discharge, on December 30. He then began to complain of almost constant pain in the right temporal and occipital regions, which was not relieved by medication. A few days before his final admission, on Feb. 17, 1948, there were nausea, vomiting and drowsiness. A few grand mal seizures were noted on the day before his readmission.

Examination on the last admission showed the patient to be in coma with Cheyne-Stokes breathing and cyanosis. The pulse rate was 100 and the blood pressure 105/60. There was weakness of the right side, more evident in the upper limb. The decompression was full but soft. The fundi showed hyperemic disks, temporal pallor and full veins; the right eye was turned outward, and the corneal reflexes were equally diminished. The deep reflexes were hyperactive, especially on the left; Babinski and Hoffmann signs were elicited bilaterally. There was no nuchal rigidity. The temperature was 98 F. and the pulse rate 110. The patient improved slightly after admission but complained of severe headache. His temperature rose to 102 F., and on March 21 he became progressively confused and ptosis of the left lid developed. Pronounced nuchal rigidity appeared and continued until his death, on March 24. Permission for necropsy was not obtained.

A spinal puncture on February 24 showed clear, colorless fluid with a pressure of 190 mm. of water and a total protein content of 120 mg. per 100 cc. Spinal puncture on March 21 disclosed deep yellow, fluorescent fluid with 185 crenated red blood cells and 6 white blood cells per cubic millimeter and a total protein content of 193 mg. per 100 cc. The presence of crenated red cells in the spinal fluid and the meningeal signs before death indicated probable bleeding into the subarachnoid space. The deep yellow tinge of the spinal fluid may have been due in part to fluorescein (20 cc. was administered intravenously on March 18).

Case 4.—J. S., a man aged 38, was admitted to the Morrisania City Hospital on June 4, 1946, with a history of progressive paralysis of the left extremities of a week's duration. Three weeks previously the patient had experienced a "momentary flash of light" while lifting a cash register. The next day he noted "pins and needles" sensations in the left side of the neck with radiation down the left arm to the fingers, followed by weakness of the extremity. The paralysis rapidly improved, but weakness of the fourth and fifth fingers, as well as numbness along the ulnar surface of the left arm, persisted. Two weeks before admission, after severe physical exertion, the patient again noted weakness of the left arm and "clicking" sensations in the left side of the neck on turning the head to the left. "Jerking" of the left shoulder and arm for about five minutes occurred during the preceding week. He "fainted" as these movements began to subside. The following day, on May 29, the patient had two convulsive seizures, each lasting about 90 minutes, with involvement of the left side of the face, the tongue and the left limbs but with no loss of consciousness. Subsequently there were two less severe attacks. The weakness of the left extremities progressed to complete paralysis. On the day of admission he complained of right supraorbital headache. During February 1946 a tumor in the right side of the neck, which had grown rapidly over a period of a month, was excised. Radium was used after the operation. The histologic diagnosis was melanosarcoma.

Examination on admission showed a well developed, well nourished man with an indurated and pigmented postoperative scar on the right side of the neck. There were complete paralysis of the left limbs with hyperreflexia and a defective plantar response on that side, absence of position sense in the fingers and toes on the left side, acral diminution of vibration in the left limbs and inconstant impairment of touch on the left side. The day after admission the patient became stuporous. The right pupil was dilated and irregular, and both pupils were fixed to light; the disk margins were blurred, and there was paralysis of the left lower part of the face, with complete spastic paralysis of the left extremities and a Babinski sign on the left side.

A spinal puncture on June 5 revealed bloody fluid with 50,000 red blood cells per cubic millimeter, an initial pressure of 430 mm. of water and an Ayala index of 2.6. The spinal puncture on June 27 again showed grossly bloody fluid, containing both fresh and crenated red cells, xanthochromic supernatant fluid but no tumor cells. The blood count and serologic reactions were normal. A roentgenogram of the chest on June 6 disclosed "one or two small round soft shadows in the middle third of the right lung." A roentgenogram of the skull showed enlargement of the sella turcica but no evidence of destruction.

The pulse became significantly slow, and stupor appeared on June 4. On June 6 the patient was unconscious but improved in a few days. On June 12, stereognostic and two point sensation were defective in the left hand, and a Babinski sign was elicited on the left side. On June 24 stiffness of the neck and the Kernig sign were present. On June 28 there were bilateral papilledema and severe stiffness of the neck. The patient complained of severe headache. On July 3 he became comatose and died. Permission for autopsy was not obtained.

Case 5.—S. B., a man aged 45, was admitted to the Morrisania City Hospital on Feb. 27, 1947, with a history of a seizure the previous evening and of pain in the upper abdominal region, with intolerance to fatty foods for nine months. X-ray examination at another hospital revealed gallstones, for which an operation was performed in September 1946. Immediately after the operation a member of the family noted a "hemorrhage" of the right eye. After being home for 10 days, the patient was "unable to walk straight." He then complained of defective vision on the right with occasional pain. After the gallbladder operation, he also complained of severe bitemporal headache and roaring noises in the ears. Three months before the operation he had become completely deaf. Six weeks before admission he was operated on for glaucoma of the right eye at Mount Sinai Hospital, complained of defective vision in the left eye for a few weeks and then became totally blind. During the two months before admission to the Morrisania City Hospital, the patient lost 45 to 50 pounds (20.4 to 22.7 Kg.). Four spinal punctures were done during his stay in Mount Sinai Hospital; all revealed bloody fluid. A note from the hospital indicated that the visual difficulty following the gallbladder operation was due to a retinal hemorrhage in the right eye and that a collection of melanin pigment was found in the gallbladder.

Examination at the Morrisania City Hospital on February 28 showed a deaf and blind man who was conscious, restless and resisted examination. There was severe nuchal rigidity. He was able to move all his limbs, and the deep reflexes were active and equal bilaterally. The Babinski sign was not elicited. Sensation could not be tested because of the patient's mental state. The right eye was proptosed, with a hemorrhage in the anterior chamber and intense injection of the bulbar conjunctiva. The left pupil was round, fixed and moderately dilated; the cornea appeared roughened and was analgesic. The right corneal reflex was intact. The media of the eyes were opaque, and the fundi could not be visualized. On March 1 the patient became drowsy and more resistive; there were pronounced meningeal signs. The patient's mental condition deteriorated gradually, and he alternated between delirium and stupor. Meningeal signs persisted. There was no fever, and the blood pressure remained at 130/100. The patient died on April 16.

There was no evidence of a blood dyscrasia. Blood studies showed secondary anemia. The serologic reactions of the blood and spinal fluid were negative. Alkaline phosphatase was 6.4 King-Armstrong units; acid phosphatase, 1.4 units. Spinal puncture yielded bloody fluid on four occasions. The spinal fluid pressure was 250 mm. of water; the number of red blood cells varied from 70,000 to 150,000 per cubic millimeter; the total protein content was 142 mg. per 100 cc., and the sugar content on two occasions was 49 and 23 mg. per 100 cc. No bacteria, fungi or yeast cells were demonstrated in cultures or smears of the spinal fluid. Melanin was present in the spinal fluid on two occasions. Melanuria was demonstrated on several examinations.

The presence of melanin in the cerebrospinal fluid is interesting. Moersch, Love and Kernohan [5] found no instance of melanin in the cerebrospinal fluid in melanomas of the nervous system. Wortis and Wortis [4] reported melanin in the spinal fluid in their fifth case. The melanotic pigment in the gallbladder, the presence of melanin in the cerebrospinal fluid and the subarachnoid bleeding justified the diagnosis of a melanoma within the nervous system. Autopsy was not obtained.

Case 6.—H. H., a man aged 39, was admitted to the Montefiore Hospital on Aug. 3, 1941, with a history of mental changes of about two months' duration and difficulty with speech. Three years previously a "dark spot" was noted on the outer aspect of the left foot. In September 1940 a biopsy revealed no evidence of malignancy. The wound healed poorly, and the pigmented lesion continued to grow. In November 1940 a mass appeared in the left inguinal region; the inguinal

5. Moersch, F. P.; Love, J. G., and Kernohan, J. W.: Melanoma of the Central Nervous System: Report of 34 Cases, in 19 of Which the Diagnosis was Verified by Operation or Necropsy, J. A. M. A. **115**:2148 (Dec. 21) 1940.

node was removed at the Memorial Hospital in December 1940. The histologic report was that of melanosarcoma. Shortly afterward nodules appeared in the subcutaneous tissue of the trunk and arms. In February 1941 he began to lose weight and complained of general weakness. In June 1941 mental symptoms and incontinence appeared.

On his admission, motor and sensory aphasia, bilateral papilledema, an indurated area at the site of a scar on the dorsum of the left foot and many hard nodules, about the size of peas, throughout the subcutaneous tissues were noted. A spinal puncture showed clear fluid with an initial pressure of 150 mm. of water and a total protein content of 54 mg. per 100 cc. There was no melanuria. The blood count showed a hemoglobin content of 55 per cent, 2,300,000 red blood cells and 14,000 white cells.

The patient died on August 23. Autopsy, by Dr. Leo Weiss, revealed melanotic metastases to the skin locally and to the subcutaneous tissues, heart, pericardium, lungs, liver, gallbladder, cystic and hepatic ducts, pancreas, esophagus, stomach, small intestine, kidneys, thyroid, right adrenal gland and popliteal, inguinal, axillary, supraclavicular, tracheobronchial and mesenteric lymph nodes. The brain showed a small area of blackish discoloration over the motor convolutions on the right side. A tumor was found in the region of the left middle and inferior temporal convolutions, close to the temporal pole. This tumor was extensively hemorrhagic. A nodule was also present in the left thalamus. Histologic studies by Dr. C. Davison revealed these brain nodules to be metastatic melanomas.

COMMENT

Table 1 shows the incidence of bloody or xanthochromic spinal fluid in cases of metastatic melanoma of the brain collected from the literature.[6] Cases of melanoma of the spinal cord were excluded because of the possibility that xanthochromia in such cases might be a manifestation of Froin's syndrome. That bloody or xanthochromic spinal fluid is as frequent with primary melanomas of the brain as with

6. (*a*) Bakody, J. T.; Hazard, J. B., and Gardner, J.: Pigmented Tumor of the Central Nervous System, Cleveland Clin. Quart. **17**:891, 1950. (*b*) Berblinger, W.: Ein Beitrag zur epithelialen Genese des Melanins, Arch. path. Anat. **219**:328, 1915. (*c*) Craig, W. M., and Kernohan, J. W.: Melano-Epithelioma of the Brain (Metastatic), S. Clin. North America **12**: 989, 1932. (*d*) Friedman, H. H., and Lederer, M.: Melanoblastoma, with Special Reference to Metastatic Dissemination, Am. J. Surg. **55**:88, 1942. (*e*) Garin, Plauchu and Masson.[1] (*f*) Gaté, J., and Boyer, C. E.: Néoplasme cutané avec généralisations à caractères mélaniques, Lyon méd. **149**:330, 1932. (*g*) Globus, J. H., and Meltzer, T.: Metastatic Tumors of the Brain, Arch. Neurol. & Psychiat. **48**:163 (Aug.) 1942. (*h*) Globus, J. H., and Selinsky, H.: Metastatic Tumors of the Brain: Clinical Study of 12 Cases with Necropsy, ibid. **17**:481 (April) 1927. (*i*) Grewal, J. S., and Kelly, W. E.: A Case of Cerebral Metastatic Melanoma Simulating Cerebrospinal Meningitis and Encephalitis, Psychiatric Quart. **8**:276, 1934. (*j*) Guillain, G., and Darquier, J.: Sarcome mélanique cérébral à foyers multiples, Bull. et mém. Soc. méd. hôp. Paris **50**:1788, 1927. (*k*) Kasanin, J., and Crank, R. P.: Melanoblastoma of the Leptomeninges, Arch. Neurol. & Psychiat. **30**:1178 (Nov.) 1933. (*l*) Kobro, M.: Et traumatisk opstått melanosarkom uden primaert naevus, Med. rev., Bergen **51**:58, 1934. (*m*) Lecouturier, R.; Ley, J.; Titeca, J., and van Bogaert, L.: La mélanose néoplastique cérébrocutanée: Contribution à l'étude des tumeurs malignes du système nerveux central avec naevi cutanés, J. belge neurol. et psychiat. **39**:103, 1939. (*n*) Loewenberg.[3] (*o*) Minkowski, M.: Über metastatische Hirngeschwülste, Schweiz. Arch. Neurol. u. Psychiat. **46**:41, 1941. (*p*) Mitchell, H. L.: Multiple Metastatic Tumors of the Brain from a Primary Melanosarcoma of the Neck, Atlantic M. J. **30**:162, 1926. (*q*) Moersch, Love and Kernohan.[5] (*r*) Morrison, A. W.: A Case of Melanoma of the Brain Resembling Epidemic Encephalitis, Journal-Lancet **42**:37, 1922. (*s*) Roger, H.; Mosinger, M.; Paillas, J., and Jouve, A.: Métastase cérébrale unique d'un mélanoblastome de la petite lèvre, Rev. neurol. **65**:1476, 1936. (*t*) Rothfeld, J.: "Blauer Naevus" des Gesichtes und melanosarkomatöse Metastasen im Gehirn, Nervenartz **6**:13, 1933. (*u*) Weimann, W.: Über melanotische Geschwülste in Zentralnervensystem, Ztschr. ges. Neurol. u. Psychiat. **85**:508, 1923. (*v*) Wortis and Wortis.[4]

TABLE 1.—*Metastatic Melanomas of the Brain*

Author	Spinal Fluid Findings
Bakody and others [6a]	
Case 2	Xanthochromic
Case 3	Xanthochromic
Berblinger [6b]	Normal
Craig and Kernohan [6c]	Normal 8 mo. before admission
Friedman and Lederer [6d]	Clear, colorless
Garin, Plauchu and Masson [1]	Grossly bloody
Gaté and Boyer [6f]	Bloody (no microscopic studies of brain lesions)
Globus and Meltzer [6g]	
Case 1	Xanthochromic on two taps
Globus and Selinsky [6h]	
Case 7	Xanthochromic
Case 8	Presumably clear
Case 9	Normal
Grewal and Kelly [6i]	Thick, cloudy fluid; 800 cells per cu. mm., predominantly polymorphonuclear leukocytes
Guillain and Darquier [6j]	Clear, colorless
Kasanin and Crank [6k]	Clear
Kobro [6l]	Bloody
Lecouturier and others [6m]	Clear
Loewenberg [6n]	
Case 2	Yellowish with brownish tinge and many red blood cells on two occasions
Case 3	Colorless
Case 4	Yellowish
Case 5	Colorless
Case 6	Very bloody
Case 7	Slightly bloody
Minkowski [6o]	Colorless
Mitchell [6p]	Yellowish
Moersch, Love and Kernohan [6q]	
3 cases	Normal except for presence of 67 lymphocytes in 1 case
Morrison [6r]	Normal
Roger and others [6s]	Clear
Rothfeld [6t]	Clear; many red blood cells; 7 lymphocytes per cu. mm.
Weimann [6u]	
Case 1	Normal
Case 2	Normal
Wortis and Wortis [4]	
Case 1	Bloody, melanotic cells
Case 2	Xanthochromic
Case 3	Slightly xanthochromic

TABLE 2.—*Primary Melanomas of the Brain*

Author	Spinal Fluid Findings
Arnvig and Christensen [7a]	Xanthochromic fluid
Bailey [7b]	Normal
Cardona [7c]	Normal
Ehnmark and Jacobowsky [7d]	Presumably clear
Farnell and Globus [7e]	Xanthochromic fluid on repeated tests
Foot and Zeek [7f]	
Case 1	Bloody
Case 2	Clear
Garcin and others [7g]	Clear on three examinations
Hesse [7h]	Serologic reactions negative (color presumably normal)
Jacob [7i]	Markedly xanthochromic; many red blood cells
Kawashima [7j]	Dark yellow, with red blood cells and leukocytes
Kraft [7k]	Two cisternal fluids clear, colorless
Mackay and Hurteau [7l]	Clear; 300 lymphocytes
Matzdorff [7m]	Bloody with supernatant xanthochromia on repeated punctures
Netherton [7n]	Yellowish on three punctures
Oliveras de la Riva and Nerin Mora [7o]	Rose-colored fluid; abundant red blood cells; 64 white blood cells per cu. mm.; supernatant fluid xanthochromic
Roger and Paillas [7p]	Clear
Schmid [7q]	Clear, colorless; 63 mononuclear cells
Shapiro and Kellert [7r]	Bloody or xanthochromic on 52 punctures
Omodei-Zorini [7s]	Many red blood cells

metastatic melanomas of the brain is shown by the cases cited in table 2.[7] The tables indicate that in 16 of the 35 proved cases of metastatic melanoma of the brain and in 10 of the 21 proved cases of primary melanoma of the brain the spinal fluid was bloody or xanthochromic, a total of 26 of 56 cases (46 per cent).

To determine whether bloody or xanthochromic fluid is commoner with metastatic melanoma to the brain than with other metastatic tumors of the brain, 56 cases of verified metastatic neoplasms at the Montefiore Hospital were reviewed. Table 3 summarizes these data.

In two of 56 cases the spinal fluid was bloody or xanthochromic. The first was that of a man aged 59 with a primary carcinoma of the lung. The spinal fluid

TABLE 3.—*Presence of Bloody Spinal Fluid in Cases of Metastatic Brain Tumors at Montefiore Hospital*

Primary Tumor	No. of Cases	Bloody Spinal Fluid
Carcinoma of the lung	25	1
Carcinoma of the breast	14	..
Hypernephroma	6	1
Nasopharyngeal tumor	3	..
Cystadenocarcinoma of the ovary	2	..
Thyroid adenocarcinoma	1	..
Sarcoma of the pancreas	1	..
Embryonal sarcoma	1	..
Adenocarcinoma of the salivary gland	1	..
Testicular tumor	1	..
Endothelioma of lymph node	1	..
	56	2

7. (*a*) Arnvig, J., and Christensen, E.: Primary Benignant Intracranial Melanoma, Acta chir. scandinav. **82**:218, 1939. (*b*) Bailey, P.: Intracranial Tumors, Springfield, Ill., Charles C Thomas, Publisher, 1933, p. 152. (*c*) Cardona, F.: Sulla melanoblastomatosi meningea, Cervello **23**:1, 1947. (*d*) Ehnmark, E., and Jacobowsky, B.: Ein Fall von meningealem Melanom mit reflektorischer Pupillenstarre, Upsala läkaref. förh. **31**:565, 1926. (*e*) Farnell, F. J., and Globus, J. H.: Primary Melanoblastosis of the Leptomeninges and Brain, Arch. Neurol. & Psychiat. **25**:803 (April) 1931. (*f*) Foot, N. C., and Zeek, P.: Two Cases of Melanoma of the Meninges with Autopsy, Am. J. Path. **7**:605, 1931. (*g*) Garcin, R.; Bertrand, I.; Thevenard, A., and Schwob, R. A.: Sur un cas de mélanoblastome diffus primitif descentus nerveux: Étude anatomoclinique, Rev. neurol. **60**:828, 1933. (*h*) Hesse, W.: Ein Fall von Melanose und primären Chromatophoromen der Meningen, Beitr. path. Anat. **71**:705, 1922-1923. (*i*) Jacob, H.: Diffus melanotische Geschwulstbildungen der weichen Hirnhäute, Deutsche Ztschr. Nervenh. **133**:167, 1934. (*j*) Kawashima, K.: Über ein Sarkom der Dura mater spinalis und dessen Dissemination im Meningealraum mit diffuser Pigmentation der Leptomeningen, Arch. path. Anat. **201**:297, 1910. (*k*) Kraft, J.: Über primäre diffuse Melanosarkomatose der weichen Hirn- und Rückenmarkhäute, Ztschr. Krebsforsch. **29**:74, 1929. (*l*) Mackay, F. H., and Hurteau, E. F.: Primary Melanoma of the Central Nervous System, J. Nerv. & Ment. Dis. **96**:369, 1942. (*m*) Matzdorff, P.: Beiträge zur Kenntnis diffuser Hirnhautgeschwülste mit besonderer Berücksichtigung der Melanome, Ztschr. ges. Neurol. u. Psychiat. **81**:263, 1923. (*n*) Netherton, E. W.: Extensive Pigmented Nevus Associated with Primary Melanoblastosis of Leptomeninges of Brain and Spinal Cord: Report of a Case, Arch. Dermat. & Syph. **33**:238 (Feb.) 1936. (*o*) Oliveras de la Riva, C., and Nerin Mora, E.: Contribución al estudio de los melanoblastomas meníngeos primitivos con la aportacíon de un caso asociado a microadenoma basófilo hipofisario asintomático, Med. clín., Barcelona **8**:19, 1947. (*p*) Roger, H., and Paillas, J.: À propos de cinq observations de tumeurs cérébrales métastatiques, Rev. neurol. **69**:730, 1938. (*q*) Schmid, H. J.: Ein Fall von primärem Melanom im Rückenmark, Frankfurt. Ztschr. Path. **33**:372, 1926. (*r*) Shapiro, I., and Kellert, E.: Primary Melanoblastosis of the Meninges, New York J. Med. **37**:2096, 1937. (*s*) Omodei-Zorini, A.: Zur Kenntnis der primärem Melanocytoblastome der Pia mater, Arch. path. Anat. **250**:566, 1924.

obtained at another hospital six months before he died was bloody (nontraumatic). Two punctures performed at the Montefiore Hospital both yielded clear fluid. At necropsy a subarachnoid hemorrhage was demonstrated in both hemispheres, being chiefly on the right side. Metastatic nodules were present in the dura and vermis of the cerebellum. Microscopic examination also showed evidence of a recent hemorrhage in the pia mater. The second case was that of a man aged 55 with a hypernephroma. There was xanthochromic spinal fluid on two occasions, with a total protein content of 63 mg. per 100 cc. Autopsy revealed a small area of pachymeningitis hemorrhagica on the under surface of the dura overlying the left superior parietal gyrus and over a metastatic tumor in the frontal lobe.

The occurrence of bloody or xanthochromic spinal fluid in cases of metastatic brain tumor has been mentioned by several investigators. Merritt and Fremont-Smith [8] found blood in the spinal fluid in three of 27 cases of metastatic tumor of the brain. Globus and Sapirstein [9] stated that in six of 32 cases of metastatic brain tumor there was evidence of blood in the spinal fluid. Since the metastatic tumors were not reported in detail in these two series, we collected the first 56 cases of metastatic brain tumor from the literature in which complete autopsies were performed and in which the nature of the primary tumor was known.[10] Table 4

8. Merritt, H. H., and Fremont-Smith, F.: The Cerebrospinal Fluid, Philadelphia, W. B. Saunders Company, 1938, p. 160.

9. Globus, J. H., and Sapirstein, M.: Massive Hemorrhage into Brain Tumor: Its Significance and Probable Relationship to Rapidly Fatal Termination and Antecedent Trauma, J. A. M. A. **120**:348 (Oct. 3) 1942.

10. Globus and Meltzer.[6g] Aring, C. D.: Diseases of Nervous System, Neurological, Clinical, Pathological Conference, 1949, vol. 10, p. 249. Barré, J. A., and Paillas, J.: À propos de deux observations de tumeur cérébrale métastatique, Marseille-méd. **1**:159, 1934. Brunner, W.: Über die Häufigkeit von Gehirnmetastasen bösartiger Geschwülste, besonders des primären Lungentumors und ihre Bedeutung für die Klinik, Ztschr. ges. Neurol. u. Psychiat. **154**:793, 1936. Challiol, V.: La metastasi cerebrale del carcinoma polmonare, Gior. psichiat. e neuropat. **67**:307, 1939. Comfort, M. W.: Yellow Spinal Fluid Associated with Tumor of the Brain, Arch. Neurol. & Psychiat. **15**:751 (June) 1926. Craig, W. M.; Woltman, H. W., and Kernohan, J. W.: Metastasis to the Central Nervous System from Carcinoma of the Lung, Am. J. Cancer **36**:12, 1939. Fattovich, G., and Lenti, P.: Contributo anatomo-clinico allo studio delle metastasi cerebrali dei tumori polmonari, Note e riv. psichiat. **66**:193, 1937. Ferguson, F. R., and Rees, W. E.: Cerebro-Spinal Metastases from Unsuspected Pulmonary Carcinoma, Lancet **1**:738, 1930. Fried, B. M., and Buckley, R. C.: Primary Carcinoma of the Lungs: IV. Intracranial Metastases, Arch. Path. **9**:483 (Feb.) 1930. Gama, C.: Metastases tumorais no encéfalo e tumores do sistema nervosa produzindo metastases, Neurobiologia **12**:1, 1949. Greenfield, J. G.: On Froin's Syndrome and Its Relation to Allied Conditions in the Cerebrospinal Fluid, J. Neurol. & Psychopath. **2**:105, 1921. King, A. B., and Ford, F. R.: A Clinical and Anatomical Study of Neurological Conditions Resulting from Metastases in the Central Nervous System Due to Carcinoma of the Lung: Review of 100 Cases, Bull. Johns Hopkins Hosp. **70**:124, 1942. Moore, M. T.: Metastatic Malignancy of the Brain and Spinal Cord: A Clinico-Pathologic Study, South. Med. & Surg. **109**:97, 1947. Pass, K. E.: Zur Klinik der cerebralen Carcinommetastasen, Nervenarzt **11**:385, 1938. Putschar, W.: Zur Pathologie und Symptomatologie der Carcinommetastasen der Zentralnervensystem, mit besonders Berücksichtigung der Hirn- und Rückenmarksnerven, Ztschr. ges. Neurol. u. Psychiat. **126**:129, 1930. Reich, J.: Über Gelbfärbung der Cerebrospinalflüssigkeit, Mitt. Grenzgeb. Med. u. Chir. **25**:721, 1913. Rimay, R. S.: Tumores metastaticos cerebrales: Estudio anatomoclinico sobre veinte observaciones, Arch. argent. neurol. **20**:89-171, 1939. Tom, M. I.: Metastatic Tumors of Brain, Canad. M. A. J. **54**:265, 1946. Sjögren, V. H.: Tumeurs intracraniennes métastatiques, Acta psychiat. et neurol. **5**:269, 1930. Winkelman, N. W., and Eckel, J. L.: Metastatic Carcinoma of the Central Nervous System, J. Nerv. & Ment. Dis. **66**:133, 1927.

summarizes these data. In six of the 56 cases, bloody or xanthochromic fluid was found; in all 6 cases, metastases from primary cancer of the lung were present.

TABLE 4.—*Bloody and Xanthochromic Spinal Fluid in Cases of Metastatic Tumor to the Brain from the Literature*

Site of Primary Tumor	No. of Cases	Bloody Spinal Fluid
Lung	36	6
Breast	5	..
Kidney	4	..
Adrenal gland	2	..
Chorioepithelioma	2	..
Colon	2	..
Ovary	2	..
Liver	1	..
Pancreas	1	..
Rectum	1	..
	56	6

SUMMARY AND CONCLUSIONS

In five of six cases of melanoma of the brain the spinal fluid was bloody or xanthochromic. The diagnosis of melanoma of the brain was confirmed at autopsy in three cases and at operation in one case. A review of the literature showed the presence of bloody or xanthochromic spinal fluid in 10 of 21 cases of primary melanoma of the brain and in 16 of 35 cases of metastatic melanoma of the brain, making a total of 26 of 56 cases, or 46 per cent.

With most other metastatic tumors to the brain, such spinal fluid findings are rare. Of 56 cases of metastatic brain tumors other than melanoma collected at the Montefiore Hospital, the spinal fluid was bloody or xanthochromic in only two. In six of 56 cases of metastatic tumors collected from the literature the fluid was bloody or xanthochromic.

Subarachnoid hemorrhage occurs more frequently with melanoma than with other metastatic tumors of the brain.

The Department of Pathology of the Montefiore Hospital, Dr. H. Zimmerman, Director, permitted use of the autopsy reports.

Abstracts from Current Literature

Physiology and Biochemistry

ANTISTREPTOLYSIN CONTENT OF CEREBROSPINAL FLUID. R. LUNDSTRÖM and G. VON REIS, Am. J. Dis. Child. **79**:438 (March) 1950.

The view usually held is to the effect that in healthy human beings and animals antibodies do not, as a rule, pass from the serum into the cerebrospinal fluid.

The present investigation comprised 159 cases in which the titer of antistreptolysin O was determined in specimens of the blood and of the cerebrospinal fluid taken at the same time. The material was divided into a group with normal cerebrospinal fluid (110 cases) and another with pathologic changes in the cerebrospinal fluid (49 cases).

Lundström and von Reis found that in persons with normal cerebrospinal fluid and with an antistreptolysin O titer of the serum of less than 200 units the titer of spinal fluid was as a rule less than 0.7 unit. The highest figure registered in this group was 1.72 units. When the antistreptolysin content of the serum rose, that of the spinal fluid also rose. Increased titers were found mainly in spinal fluids showing pathologic changes. The highest value (25 units) was found in two cases of purulent meningitis.

ALPERS, Philadelphia.

SPINAL FLUID IN ACUTE POLIOMYELITIS: CHANGES IN TOTAL PROTEIN AND CELL COUNTS ON SERIAL STUDY. G. D. FORD, F. L. ELDRIDGE and C. G. GRULEE, Am. J. Dis. Child. **79**:633 (April) 1950.

One hundred and forty-two serial examinations of the spinal fluid were made on 31 patients with acute poliomyelitis. Ford and his associates found that the peak protein values were reached by the fourteenth day of illness in most patients and that consistently higher values were obtained in patients with paralysis.

A significant decrease in the total number of cells occurred after the first period (one to four days) in the paralytic patients, whereas a similar decrease occurred after the second period (five to eight days) in the nonparalytic patients. In both the nonparalytic and the paralytic patients there was a gradual shift in cell dominance. Early in the course of the disease there was approximately equal distribution between mononuclear and polymorphonuclear cells, and after the first week there was predominance of mononuclear cells.

ALPERS, Philadelphia.

ELECTRONARCOSIS: IV. EFFECTS OF SQUARE WAVE APPLICATION TO MOTOR AREA FOLLOWING DESTRUCTION OF DIENCEPHALON. E. MARTINI, T. GUALTIEROTTI and A. MARZORATI, J. Neurophysiol. **13**:113, (March) 1950.

After the diencephalic center has been destroyed by diathermy, stimulation of the cortex with interrupted current is without effect. After a few minutes the normal electrical activity of the cortex slowly decreases, and the cortex is insensible to electrical, mechanical or chemical stimuli.

The phenomena may be interpreted as due either to (*a*) fatigue of cellular elements of the cortex, whose activity is not controlled by the inhibitory diencephalic center, or to (*b*) random activity such that, the number of neurons in question being very great, the algebraic sum of their electrical manifestations tends to equal zero.

Electrical silence follows passage of the interrupted current, whatever may be the position of the recording electrodes on the skull.

After this stage electrical activity is resumed, being characterized first by very rapid waves, which later increase in amplitude and appear in groups. During this period the patient has mental confusion and often catatony.

ALPERS, Philadelphia.

ELECTRONARCOSIS: V. FARADIC STIMULATION OF MOTOR AREA FOLLOWING DIENCEPHALIC DIATHERMY, J. GUALTIEROTTI, E. MARTINI and A. MARZORATI, J. Neurophysiol. **13**:117 (March) 1950.

If the motor cortex is stimulated with an inductorium instead of with an interrupted current, wave potentials of the strychnine type appear, and partial inhibition, similar to that obtained with the interrupted current, occurs.

In an animal in which the diencephalic center has been destroyed, faradic stimulation of the motor cortex is without effect; on the contrary, strychnine solutions applied to the cortex give rise to the appearance of strychnine waves. The application of interrupted current in the presence of strychnine does not produce inhibition, as it does when the diencephalic center is intact, but results only in a local excitation.

The authors believe that they have demonstrated a center extending from the frontal part of the diencephalon back to the pons. This center has a regulatory action on all parts of the central nervous system, from the telencephalon to the spinal cord; it has a segmentary structure, and the segments may be independently stimulated both directly or reflexly.

ALPERS, Philadelphia.

COURSE OF SPINOTHALAMIC AND MEDIAL LEMNISCUS PATHWAYS IN CAT AND RHESUS MONKEY. C. M. BERRY, R. C. KARL and J. C. HINSEY, J. Neurophysiol. **13**:149 (March) 1950.

Stimulation of the sciatic and saphenous nerves of the cat and rhesus monkey initiated potentials in the medulla oblongata, midbrain, thalamus and internal capsule which were characterized by an initial rapidly conducted spike, followed by slower spikes.

A large portion of the afferent pathways from the saphenous nerve passed lateral to the inferior olivary nucleus. The afferent impulses from both the sciatic and the saphenous nerve traversed the midbrain in a restricted area corresponding to the medial lemniscus and then entered the nucleus ventralis posterolateralis. Impulses following stimulation of the sciatic and saphenous nerves ascended in the same relative positions on the two sides of the brain stem. The component of the medial lemniscus from the nucleus gracilis and nucleus cuneatus crossed completely to the opposite medial lemniscus and thalamus.

ALPERS, Philadelphia.

CLASSIFICATION AND SIGNIFICANCE OF ELECTROENCEPHALOGRAPHIC PATTERNS PRODUCED BY NITROUS OXIDE-ETHER ANESTHESIA DURING SURGICAL OPERATIONS. R. F. COURTIN, R. G. BICKFORD and A. FAULCONER JR., Proc. Staff Meet., Mayo Clin. **25**:197 (April 12) 1950.

This investigation was undertaken to ascertain (1) whether the changes in the electroencephalographic patterns occurring under nitrous oxide-ether anesthesia were sufficiently consistent to be classified and (2) whether these changes could be used as a practical measure of depth of anesthesia.

Courtin and his co-workers used a specially designed electroencephalograph for recording the electroencephalogram and electrocardiogram in the operating room.

The investigation of 45 patients receiving ether anesthesia showed that there is a constant relation of the electroencephalographic pattern to the depth of nitrous oxide-ether anesthesia.

The electroencephalographic patterns associated with deepening anesthesia can be conveniently classified into seven electroencephalographic levels of anesthesia. The authors describe use of these levels as a guide to practical anesthesia.

ALPERS, Philadelphia.

Psychiatry and Psychopathology

HYPERSOMNIA SYNDROMES. M. N. PAI, Brit. M. J. **1**:522 (March 4) 1950.

Pai made a study of 67 male patients who complained of either diurnal somnolence, which Pai defines as "drowsiness during which a patient is partially aware of what is happening around him," or hypersomnia, a term used to mean "a condition in which daily more hours are spent in sleep than in wakefulness." Psychiatric investigation revealed that most of the patients had immature and inadequate personalities and had previously reacted to stress with the production of psychogenic symptoms. Psychologic and environmental factors were found to be responsible for the symptoms in every case. Excessive sleep and abnormal appetite in the same patient, both neurotic manifestations, were found to be due to two entirely different factors.

The case is reported of a man aged 20 who slept from 7:30 p. m. until 2:30 p. m. the next day. Examination indicated that during these 18 hours of "sleep" he was actually asleep only about 4½ to five hours; the rest of the time he was in a state of partial sleep, simulating a hypnotic trance. The psychologic factors were discovered by means of hypnosis, and the patient was succesfully treated. After three weeks he was able to awaken in the morning without any trouble, and he has had no difficulty in the five years since then.

ECHOLS, New Orleans.

Diseases of the Brain

RHEUMATIC BRAIN DISEASE AS A CAUSE OF CONVULSIONS. J. F. WHITMAN and L. J. KARNOSH, Cleveland Clin. Quart. **16**:136 (July) 1949.

Whitman and Karnosh report on five patients with histories of recurrent generalized convulsive seizures, for several of whom a previous diagnosis of idiopathic epilepsy had been made. Each patient had clinically demonstrable rheumatic heart disease. None had convulsions before the age of 34. Three had a past history of definite migratory polyarthritis or chorea; their convulsive seizures, however, did not begin until 10, 12 and 32 years later.

Of the criteria for the diagnosis of rheumatic brain disease prior to necropsy, the clinical demonstration of rheumatic heart disease is imperative and a history of acute rheumatic fever is desirable. Electroencephalography appears to be of no specific diagnostic value; the records in the cases reported were abnormal but showed no deviation which might be considered in any way pathognomonic of the lesion.

An accurate history stressing time relationship is of decided importance. Knowledge of the age of the patient at the time of onset of symptoms is a valuable aid. The latent period between the active polyarthritis and the appearance of symptoms indicating involvement of the central nervous system is observed so commonly that its significance cannot be doubted, although its explanation is not clear.

A conservative consideration of rheumatic brain disease, along with other causes of convulsions, is desirable.

ALPERS, Philadelphia.

Peripheral and Cranial Nerves

MÉNIÈRE'S DISEASE: A PROPOSED CLASSIFICATION AND DIFFERENTIAL DIAGNOSIS. B. RACHLIS, Arch. Otolaryng. **52**:373 (Sept.) 1950.

In view of the different sites of origin of the triad of symptoms of vertigo, tinnitus and deafness, which may occur singly or in combination; and in view of the varying etiology, the author proposes that Ménière's syndrome be classified as peripheral, central or psychosomatic. The term "Ménière's disease" should be dropped, since it does not constitute a definite pathologic entity.

The principal diagnostic points of differentiation between a peripheral and a central lesion are the periodicity of attacks, freedom from symptoms between attacks and the presence of cochlear symptoms, such as tinnitus and fluctuating impairment of hearing (first of the conduction type and later of the mixed conduction and perception type), in addition to the combined proportionate association of vertigo, past pointing, nystagmus falling in acute attacks, presence of vestibular test findings suggestive of a peripheral lesion, and absence of positive neurologic signs and symptoms.

ALPERS, Philadelphia.

SYNDROME OF BILATERAL VESTIBULAR PARALYSIS AND ITS OCCURRENCE FROM STREPTOMYCIN THERAPY. P. NORTHINGTON, Arch. Otolaryng. **52**:380 (Sept.) 1950.

The disturbance of equilibrium caused by bilateral loss of vestibular function from streptomycin therapy appears to be basically similar to that of bilateral vestibular paralysis from other causes, whether the site of the lesion is in the peripheral or the central components of the vestibular nervous system.

Fifty-six patients were observed who experienced symptoms suggestive of bilateral vestibular paralysis from the toxicity of streptomycin. Six of the patients had other pathologic conditions to account for their disabilities. In only one did streptomycin cause impairment of hearing sufficient for it to be detected by the patient. All patients receiving a daily dose of 2.5 to 3.2 Gm. (37.5 to 50 grains) of streptomycin had neurotoxic effects of the drug, which usually occurred during the second week of its administration. Fifty patients were observed who received a daily dose of 1.0 Gm. (15 grains) of streptomycin for 75 days; only one experienced neurotoxic effects.

The regular impairment of the vestibular-ocular reflex and the occasional impairment of the cochlear reflex in patients with neurotoxic symptoms due to use of streptomycin permit the exclusion of the cerebellum as its chief focal lesion.

Although it is customary to observe a good degree of compensation after total and permanent loss of the aid of afferent impulses of any one of the sensory nervous systems contributing to equilibratory function, there may be a severe disturbance of equilibrium due to streptomycin without total loss of even one sensory function, the vestibular. A good degree of compensation has been noted to occur early in some patients in the presence of gross loss of reactions in vestibular tests and slowly in others when such reactions were relatively good. Northington concludes, therefore, that the coordinating and integrating processes of afferent impulses into terms of muscular activities are damaged by streptomycin and that intactness of the central vestibular components is of greater importance to maintenance of equilibrium than normal afferent sensory systems.

ALPERS, Philadelphia.

A CLINICAL CLASSIFICATION OF VESTIBULAR DISORDERS WITH DIFFERENTIATION OF THREE SYNDROMES AND DISCUSSION OF A COMMON FORM OF VERTIGO INDUCED BY SUDDEN MOVEMENT OF THE HEAD. F. R. FORD, Bull. Johns Hopkins Hosp. **87**:299 (Oct.) 1950.

Vertigo is a common and distressing symptom, but there is no general agreement about the interpretation of even the types which occur most frequently. Ford separates the disorders of the vestibular apparatus into three chief syndromes: (*a*) those due to spontaneous discharge, (*b*) those due to increase of vestibular reflexes and (*c*) those due to loss of vestibular reflexes.

The first syndrome, i. e., symptoms produced by spontaneous discharge, is observed in an isolated form in Ménière's disease. The same symptoms may occur in acute processes involving the labyrinth or the eighth nerve, such as labyrinthitis of various types, vascular disorders, intoxications, trauma and, perhaps, allergic reactions. In cases of this type the picture is complicated for a time by increase in reflexes, and eventually by loss of reflexes.

The second syndrome, i. e., symptoms due to increase of vestibular reflexes, is seen not only in association with labyrinthitis and with various types of vestibular irritation, as mentioned above, but much more commonly in subjects of middle age who have been subjected to prolonged

nervous strain. In such patients the symptoms are usually transient and there is no deafness or loss of function. This condition has been termed "postural vertigo," but since the vertigo is induced by movement of the head it might well be termed "movement vertigo." In elderly patients these symptoms are very common and are apt to be persistent.

The third syndrome, i. e., symptoms due to loss of vestibular function, may result from any destructive process involving the vestibular apparatus, whether acute or slowly progressive. When one labyrinth or one eighth nerve is involved, the symptoms are usually of only a few months' duration, for compensation eventually occurs; but when both nerves are involved, the symptoms are persistent. Ford points out that is is important to distinguish between discharge and loss of function, so as to avoid useless operations.

ALPERS, Philadelphia.

NON-PENETRATING INJURIES OF THE MEDIAN NERVE AT THE WRIST. D. KENDALL, Brain **73**:84, 1950.

Kendall reports on 14 cases of medial nerve paralysis. In eight cases, the condition was unilateral, and the nature of the trauma, usually handling of implements in everyday use without awareness of injury, was clear. In only two of these six cases was there evidence of complete interruption of conduction. When the interruption was incomplete, motor symptoms were slight, but sensory, vasomotor and trophic changes were prominent. Of the six cases in which the trauma was bilateral, no definite history of injury could be obtained in five. This group consisted of middle-aged women who were engaged in the performance of heavy housework and laundry; the condition resulted from normal activity of the hands. In this group, motor loss tended to be severer, and trophic disturbance was present and bore a definite relation to the degree of loss of sensation.

In six of the eight cases of unilateral paralysis the condition was mild and responded well to immobilization of the wrist and protection of the palm. In all the six cases of bilateral paralysis the disorder was severe and was treated surgically by division of the transverse carpal ligament. It is believed that friction neuritis is produced in the median nerve in the carpal tunnel, where it is compressed between the flexor tendons, posteriorly, and the carpal ligament and palmaris longus, anteriorly.

FRANKEL, Philadelphia.

DIABETIC NEUROPATHY AND HEPATOMEGALY. R. N. HERSON, Proc. Roy. Soc. Med. **41**:104 (Feb.) 1948.

Herson reports the case of a patient aged 64 with a history of diabetes of ten years' duration and irregular treatment. He presented moderate hypertension, arteriosclerosis, hepatomegaly and immature cataracts. The pupils did not react to light or in accommodation. The left foot was gangrenous, and the leg reflexes were absent. Eight months later there developed ptosis bilaterally, almost complete internal and external ophthalmoplegia and corneal reflexes. All tendon reflexes were absent except for the right biceps. Pain sensation was absent below the knees, but touch sense was preserved. Vibration sense was absent, but position sense was present. There was no evidence of syphilis.

BERRY, Philadelphia.

MÉNIÈRE'S ATTACK. HANS BRUNNER, Confinia neurol. **10**:1, 1949.

Brunner believes that the customary concept that the Ménière attack consists of dizziness, deafness and tinnitus is incomplete. He reasserts the dictum that the genuine Ménière attack is invariably associated with spontaneous nystagmus, usually of a high degree of intensity. He distinguishes four types of Ménière attacks: grand, partial and abortive attacks and Ménière's equivalents.

The grand attacks are those which consist of "objective and subjective labyrinthine . . . and cochlear symptoms." They may be sufficiently intense to produce loss of consciousness. The partial attacks are those which consist of only labyrinthine or only cochlear symptoms.

The author discusses at some length his reasons for accepting the concept of "strictly cochlear attacks," in Ménière's disease. Abortive attacks are those "minor attacks of dizziness or increased tinnitus and deafness which frequently occur and usually do not alarm the patient." Ménière's equivalents are those in which the leading symptom, viz., vertigo, "is not prominent or may even be absent, and is replaced by subjective symptoms of another type, viz., headache or gastrointestinal symptoms," but always with "nystagmus of the highest intensity."

The author disagrees with those who assert that Ménière's attacks must necessarily cause permanent impairment of hearing. "A patient's hearing may be permanently lost after a few attacks or even after the first noticeable attack or it may be only slightly damaged after a great number of attacks."

FOLEY, Boston.

Treatment, Neurosurgery

THE HOSPITAL TREATMENT OF DEMENTIA PRAECOX: II. E. A. ELLISON and D. M. HAMILTON, Am. J. Psychiat. **106**:454 (Dec.) 1949.

Ellison and Hamilton studied 100 men with dementia precox. Besides the usual therapeutic procedures, including intensive psychotherapy, physical education, occupational activity, physical therapy and a program of socialization, all the patients received electric shock treatment. Repeated series of shock treatment were given to more than one third, with an interval of about one month between each series.

Of the 100 patients, 57 per cent are at home and improved at the end of a follow-up period of two to eight years. Thirty-eight are considered recovered or much improved. Six of the seven hebephrenic patients are unimproved, while one is at home, improved. Two patients in the category of simple dementia precox are recovered; two are at home improved, while two are unimproved.

A comparison of this study with a previously reported study of 100 women patients reveals several interesting differences. The ultimate response as a group to treatment was noticeably better in the group of women than in that of the men. In the catatonic group, 20 per cent more women than men were substantially benefited, and in the paranoid group a 10 per cent increase was noted.

The prepsychotic personalities in both groups were predominantly of the passive type. In their illness, however, the women tended to unleash their aggressive drives, while the men showed an increased passivity, often amounting to an undisguised plea for emasculation. The cultural roles of the two sexes were thus reversed at the time of personality breakdown.

Ellison and Hamilton point out that our culture demands of men an aggressive reaction to life and generally has little tolerance of the individual male who expresses great need for passivity and dependence. This is not so obvious in the case of women. Our culture accepts both the passive, dependent woman and the aggressive woman with masculine strivings. It is this which possibly accounts for better results among women patients with dementia precox.

ALPERS, Philadelphia.

ROENTGEN THERAPY OF CUSHING'S SYNDROME (PITUITARY BASOPHILISM): REPORT OF A CASE OBSERVED FOR ELEVEN YEARS. E. A. POHLE, and I. WEISSMAN, Ann. Int. Med. **32**:542 (March) 1950.

The case reported by Pohle and Weissman is unique in that, until recurrence of symptoms, the primary therapeutic measure was irradiation directed to the pituitary gland. Remission of the disease followed for a period of about eight years. The authors emphasize that improvement did not occur after the first series of x-ray treatments but that definite response to treatment was noted after the fourth series. After the fifth series the symptoms regressed to the point where further treatment was no longer indicated. The authors also emphasize the importance of an adequate dosage if good results are to be expected.

When symptoms recurred after the remission, hypophysectomy was attempted. The patient had an uneventful postoperative course but showed no definite change in his symptom complex. Shortly after repair of an abdominal incisional hernia was attempted, secondary infection of the wound occurred, and the patient died.

The presence of fibrosis and atrophy of the pituitary gland noted at the time of hypophysectomy and at necropsy is evidence of the ability of roentgen radiation to eliminate hyperfunction of this gland. It is conjectural, but reasonable, to assume that the recrudescence of symptoms in this case may have been due to the development of compensatory hyperplasia of the adrenal glands.

ALPERS, Philadelphia.

TRACHEOTOMY IN POLIOMYELITIS. R. J. STROBEL and N. CANFIELD, Arch. Otolaryng. **52**:341 (Sept.) 1950.

Strobel and Canfield discuss the indications for tracheotomy under two heads: 1. Therapeutic tracheotomy should be considered whenever (*a*) any respirator patient exhibits signs of bulbar involvement or displays persistent cyanosis or struggling (restlessness and combativeness usually mean air hunger), or (*b*) any nonrespirator patient is unable adequately to keep his airway free of secretions, as evidenced by moist rales, choking and cyanosis.

2. Prophylactic tracheotomy should be seriously considered whenever (*a*) early and progressive bulbar signs are present and before the patient has difficulty, and (*b*) a long siege in the respirator seems probable, even though no bulbar signs are present. A patient with severe respiratory paralysis may have difficulty in handling pharyngeal mucus because of inability to cough effectively.

Tracheotomy was performed on 10 patients with bulbar poliomyelitis. Five of the patients died. In all surviving tracheotomized patients the tracheotomy was probably a life-saving procedure. Earlier tracheotomy for three of the five who died might have been life saving. The authors state that the otolaryngologist should be consulted immediately concerning every patient with poliomyelitis who shows symptoms of bulbar involvement or respiratory difficulty.

ALPERS, Philadelphia.

RELIEF OF PAIN IN CHRONIC RELAPSING PANCREATITIS BY UNILATERAL SYMPATHECTOMY. A. HURWITZ and J. HURWITZ, Arch. Surg. **61**:372 (Aug.) 1950.

Visceral afferent impulses from the pancreas travel through the splanchnic nerves and lower thoracic sympathetic chain. Pain produced by chronic pancreatitis therefore can be relieved by low thoracic sympathectomy (seventh to twelfth thoracic) and splanchnicectomy. In most instances unilateral sympathectomy on the side of pain is satisfactory. In some cases the bilateral operation is necessary, particularly when the head of the pancreas is involved.

LIST, Grand Rapids, Mich.

TREATMENT OF NEURONITIS WITH BAL. J. M. NIELSEN, Bull. Los Angeles Neurol. Soc. **15**:61 (June) 1950.

Ten cases of the Guillain-Barré syndrome treated with dimercaprol injection U. S. P. (BAL) are reported. The only criterion for selection of cases was polyneuritis with albuminocytologic dissociation in the spinal fluid and considerable increase of total protein above the normal, or polyneuritis with facial diplegia.

In nine cases benefit was derived from the use of dimercaprol, as shown by improvement when it was given and cessation of improvement when it was discontinued. In several cases there was immediate, but unsustained, improvement. In one case, that of a diabetic, there was no improvement.

While this work is only experimental, it seems to show that the Guillain-Barré syndrome is basically a manifestation of a toxic interference with enzyme metabolism of ganglion cells, which can be aided with dimercaprol. However, the optimum dose is unknown, and it seems to vary not only with the etiologic factor but also in the same case at various times. The dose used by Nielsen was 2.5 mg. per kilogram of body weight, which was reduced or increased as circumstances required. There was a suggestion in several cases that transient bulbar and respiratory difficulties appear with the use of the drug.

ALPERS, Philadelphia.

USE OF PITUITARY ADRENOCORTICOTROPIC HORMONE (ACTH) IN POLIOMYELITIS. L. L. CORIELL, A. C. SIEGEL, C. D. COOK, L. MURPHY and J. STOKES JR., J. A. M. A. **142:** 1278 (April 22) 1950.

Certain data on poliomyelitis suggest an unusual host-parasite relationship. Many observations indicate that susceptibility or resistance to poliomyelitis involves nonspecific defensive mechanisms and, indirectly, that the host response is more important than the virulence of the virus in determining the course of infection. Rapid mobilization of bodily defensive mechanisms may limit the spread of infection in the usual nonparalytic case, whereas a slower response as a result of constitutional factors or exhaustion may permit infection to spread. It is not known whether or not poliomyelitis precipitates an "alarm reaction." Pituitary adrenocorticotropic hormone (ACTH) induces this reaction quickly. It occurred to the authors that a failure of these mechanisms to act naturally might contribute to the development of paralytic poliomyelitis.

The therapeutic use of pituitary adrenocorticotropic hormone was observed in a group of 70 patients who had been ill less than five days, 35 receiving pituitary adrenocorticotropic hormone and 35 a placebo.

It was found that the "alarm reaction" is mobilized in poliomyelitis, as shown by the eosinophil response. In these patients, pituitary adrenocorticotropic hormone produced a physiologic effect, as evidenced by a further depression of the eosinophil count and by increased excretion of 17-ketosteroids. There was no demonstrable effect in the treated as compared with the untreated group when evaluated on the basis of (1) temperature response, (2) paralysis, (3) progression of paralysis or (4) early residual effects.

Coriell's statistical analysis shows clearly that pituitary adrenocorticotropic hormone has no beneficial or obvious deleterious effect on the course of poliomyelitis when treatment is begun after the onset of symptoms.

ALPERS, Philadelphia.

AUREOMYCIN TREATMENT OF MENINGITIS DUE TO BACILLUS PYOCYANEUS AND BACILLUS AEROGENES. E. NETER, R. F. KRAUSS, G. J. EGAN and T. H. MASON, J. A. M. A. **142:** 1335 (April 29) 1950.

Acute purulent meningitis due to Bacillus pyocyaneus (Pseudomonas aeruginosa) of the melanogenic type and meningitis due to Bacillus aerogenes (Aerobacter aerogenes), respectively, were observed in two children as complications of surgical exposure of the spinal cord. In both instances the infection entered the meninges through a cerebrospinal fluid leak and occurred during prophylactic administration of penicillin. Both strains were susceptible to aureomycin, though the strain of B. aerogenes was extraordinarily resistant to streptomycin. It was felt that recovery from the meningitis could be attributed largely to aureomycin therapy.

The patient with meningitis caused by B. pyocyaneus received the drug orally, rectally and intravenously. Aureomycin was given to the other patient orally, rectally, intrathecally and intraventricularly. No immediate ill effect was noticed from the intrathecal or intraventricular administration of aureomycin in the dosage employed. Neter and his co-workers believe that these routes of administration should be resorted to only if all other routes of therapy fail.

ALPERS, Philadelphia.

CLINICAL MANAGEMENT OF ACUTE POLIOMYELITIS. E. SMITH, D. J. GRAUBARD, J. FALCONE, T. B. GIVAN, P. ROSENBLATT and A. FELDMAN, J. A. M. A. **144**:213 (Sept. 16) 1950.

At present there is no form of treatment of acute poliomyelitis that limits the spread of virus in the central nervous system or alters its effect on the nerve cells. In the past few years there has been an increasing interest in the control of pain and vasomotor instability. Various means of sympatholysis have been tried to correct the vasomotor dysfunctions. Maintenance of circulation in the muscle aids in its rehabilitation, and the degree of recovery is directly related to the time of institution of corrective measures.

In the present study the objective has been to shorten the duration of pain, muscular spasm and vasomotor instability during the acute and subacute stages of poliomyelitis so that measures of rehabilitation of the patient can begin as early as possible. During 1949, 663 patients with acute poliomyelitis were treated with benzazoline (priscoline®; 2-benzyl-4,5-imidazoline) hydrochloride, a sympatholytic drug. Smith and his colleagues believe that benzazoline was a useful adjuvant in the early management of the disease. The benefits to treated patients included the relief of pain, spasm and vasomotor dysfunction. In the doses described, morbidity was minimal.

Nearly all patients manifested some form of progress, and their clinical status improved so rapidly that it was possible to transfer the majority of them, relaxed and free of pain, to their homes or to orthopedic hospitals in seven to 14 days. The nursing personnel was relieved of the tremendous patient load that they would have had if treatment with hot packs had been used. Results such as these had not been accomplished in the past with any other form of symptomatic therapy. The further investigation of benzazoline and other sympatholytic drugs is indicated.

ALPERS, Philadelphia.

FERROUS CARBONATE IN TREATMENT OF TIC DOULOUREUX. L. M. DAVIDOFF and E. H. FEIRING, Acta psychiat. et neurol. **24**:403 1949.

Davidoff and Feiring review the literature on the use of ferrous carbonate in the treatment of trigeminal neuralgia. Hutchinson (Cases of Neuralgia Spasmodica, Commonly Termed Tic Douloureux, Successfully Treated, London, 1822) appeared to have been enthusiastic about this form of therapy. The present authors evaluated this method in the treatment of 91 patients—49 men and 42 women, between the ages 23 and 79, with 85 per cent of all patients in the age range of 41 to 20 years. The average duration of illness was five years. The second and third branches of the fifth nerve were most frequently involved. Of the 91 patients, 57 reported no relief of symptoms; 34 patients, or about one third, benefited appreciably. At the time of the report, improvement had lasted from six months to six years. Recurrence of pain was noted in 17 patients, and operation was undertaken on 11 of these.

The treatment consists in the administration of 4 capsules of ferrous carbonate, each containing 1 Gm., twice daily with meals. A total of 8 Gm. daily is used, and the treatment is continued from weeks to months. Improvement appears one to two days after therapy is instituted. Side reactions to the medication include nausea, vomiting, diarrhea and constipation and may be countered with elimination of the drug for several days.

J. E. PISETSKY, New York.

Diseases of the Skull and Vertebrae

OXYCEPHALY. M. BODIAN, J. A. M. A. **143**:15 (May 6) 1950.

Oxycephaly consists of a group of congenital deformities resulting from premature closure of the bony vault of the skull. The outstanding feature is a tower-shaped head. Developmental anomalies of other parts of the body are frequently associated. Because of the numerous ocular observations, the ophthalmologist sees many of these patients.

Bodian reports a case of typical oxycephaly with optic nerve atrophy in a young Negro boy. The role of injury to the eyes of oxycephalic persons is strikingly demonstrated in this case.

ALPERS, Philadelphia.

RECURRENT ANENCEPHALY. N. FERRERLICHT, J. A. M. A. **143**:23 (May 6) 1950.

In spite of statistical evidence to the contrary, there persists general acceptance of the belief that congenital abnormalities are unlikely to recur. According to Murphy, however, a mother of a defective child is twenty-five times as likely to produce another defective offspring as is a mother in the general population. Further, Murphy showed that there is nearly one chance in two that a subsequent malformed child will exhibit the same defect as that shown by its earlier born sibling.

Ferrerlicht reports a case of recurrent anencephaly in a primigravida aged 21 at the first delivery and 23 at the second delivery. The incidence of recurring anencephaly is high. Female anencephali predominate. Generally the bodies of anencephali are well formed. In this condition the length of gestation is abnormal, and hydramnios is a common complication.

ALPERS, Philadelphia.

Society Transactions

PHILADELPHIA PSYCHIATRIC SOCIETY

Theodore Dehne, M.D., *Presiding*

Regular Meeting, Oct. 13, 1950

COMBAT NEUROSES

Lessons of World War II. COMDR. ROGER D. SHERMAN (MC), U. S. N.

Lessons, largely adduced from recent literature, were considered in terms of the following factors:

1. *Selection of Men for Service.*—The high rate of rejection, with attendant effects on morale, the fallacy in believing that induction screening would eliminate casualties and the effective combat performance of many psychoneurotic men led to the conclusion that manpower waste was tremendous. It was suggested that a continuous screening system be adopted with actual, not conjectured, failure to adjust to service as the criterion.

2. *Prevention of Military Psychoneurotic Disorders.*—The great incentive value of such short term goals as a definite tour of combat duty, the primary importance of group solidarity and identification among individual combatants, and the key role of leadership talent and emotional stability in officers were emphasized.

3. *Recognition of Combat-Induced Disability.*—The need to avoid, on the one hand, the perpetuation of symptoms by overzealous attention to mild psychological symptoms and, on the other, abrupt unintelligent dismissal of significant symptoms was accented. Except in the case of officers, initiative in recognition should be left to the individual and/or his comrades. Psychological features, as anxiety, startle, irritability and depression, and psychosomatic symptoms, particularly of a type referable to the autonomic nervous system, are well nigh universal in combat. Except when grossly incapacitating, they should be considered within the realm of normal. The incidence of psychosis in the services does not increase in wartime and is not a major problem. Inefficiency, inadequacy, ineptness and nonmotivation should not be confused with psychoneurotic syndromes.

4. *Treatment of Combat Casualties.*—The fundamental problem is restoration of personality equilibrium rather than the alteration of long-standing neurotic patterns. The contribution of prompt treatment close to the combat area, narcosynthesis and group therapy was regarded as an advance in military psychiatry.

Combat Reactions. LIEUT. COMDR. ALBERT J. ZUSKA (MC), U. S. N.

The concept of war-induced neuroses has undergone significant changes during the two world wars. The term "shell shock," used during World War I, was devastating and probably set off more neurotic reactions through its injudicious use than did the battle conditions under which the men fought. The clinical pictures of combat reactions are parallel to the neuroses of civilian life, and they show essentially the same mechanisms. Although these are somewhat modified by war conditions, the basic therapeutic principles are essentially the same as in civilian life, with the important exception that treatment must be limited in scope and must aim toward returning casualties to duty as soon as possible.

The Navy adopted the term "combat fatigue" to designate the reaction of men who show signs of fatigue after severe exposure to combat conditions for a sufficient length of time. Four arbitrary criteria were established for this diagnosis. They are (1) a stable personality prior to the appearance of the emotionally determined combat experience; (2) a combat experience of sufficient intensity to render it capable of producing the symptoms; (3) objective evidence of subjective anxiety; (4) recoverability.

The deficiencies of the term "combat fatigue" are at once recognized, but at least it is benign. A fatigue resulting from combat implies that the patient should recover after removal from combat, sufficient rest and appropriate treatment. Unlike the term "shell shock," the patient will not be able to trade on it for the remainder of his life. The term is a working diagnosis in the Navy, and the fact that no one is ever discharged from the Navy with it, keeps it intramural. Since there is doubt that this reaction is really a neurosis, the term "combat fatigue" serves a double function—it keeps the "stigma" of neurosis from a man's medical record until there has been sufficient lapse of time to study him in a hospital.

Current Korean Casualties. COMDR. LEO S. MADLEM JR. (MC), U. S. N.

The present Korean casualties were arbitrarily divided into (1) the group of two war combat veteran psychiatric casualties, (2) the group of so-called traumatic neuroses and (3) the inadequate group. The inadequate group were not discussed. In this study, the group of traumatic neuroses consisted of the men who had been overwhelmed by their combat experiences and in whom were seen strains of passive-dependent needs, hostility, aggressiveness, latent homosexual fears and other unresolved conflicts. A wire recording was played which portrayed the battle experiences of one of these men as relived under the influence of thiopental sodium. This abreaction displayed the actual situations he encountered, as well as demonstrated his feelings of isolation, his guilt, etc.

The two war combat casualty group included that group of men who had successfully performed during the late world war but who quickly succumbed to the stress of war in Korea. A case in point was presented of a man who had fought for 31 months in Europe as a machine gun section leader. He had been decorated for bravery and had been offered a battle field commission, which he had refused. He had been wounded twice. At no time during that war did he manifest symptoms suggestive of a psychoneurosis. He remained in the Army. When ordered to combat duty in Korea, he began to exhibit neurotic symptoms, and after five days of Korean combat, there developed a "dissociated state," which required his evacuation to the States. Significant factors were considered to be his increased responsibilities (marriage and children), a changed philosophy and lack of identification with the Army and his squad.

DISCUSSION OF PAPERS ON COMBAT NEUROSES

DR. THEODORE DEHNE: We have a number of well qualified discussers with us. The first of these, Dr. Strecker, is known to all of us as the head of the department of psychiatry at the University of Pennsylvania School of Medicine. He has studied these problems in their broader aspects for many years.

DR. EDWARD STRECKER: I do not intend to discuss these three excellent presentations in any detail. I should like to talk in general terms about military neuropsychiatry, particularly in its relation to morale. The better the morale, the fewer the psychiatric casualties. All military leaders knew this long before there was such a thing as military psychiatry. The greatest of them, Napoleon, said that 25 per cent of a victory was due to personnel and materiel and 75 per cent to spirit, which is just another word for morale. The great French army was broken when its morale was broken before Moscow. An army may move on its belly, but it really takes its objective by morale.

There are many other aspects. I have always felt that we too often lead men to think that when they go into battle their fear will be dispelled. It almost never is, but, rather, is increased. In fairness to themselves and everybody else, men ought to be taught to mobilize their fear, and in that way become much more effective as soldiers.

DR. LAUREN SMITH: I was interested before the second world war in the criteria of selections for the armed forces and considered myself something of an authority on how to select personnel. I have since found how inadequate my ideas were, and how distorted. These papers bring out well the extent to which we may expect a repetition of acute problems in successive wars. They particularly emphasize the necessity of having some means of finding a common denominator to stimulate the interest of every individual person in the economy of our war effort. The thing that stimulates my interest in discussions about the future Army and in getting efficiency of manpower is the question of motivation.

All branches of the armed services believe now that psychiatric examination should no longer be used as a final basis for rejection. It should not be used to throw out any except the epileptic with frequent convulsions, or the frankly psychotic or the completely inept applicant.

DR. CALVIN DRAYER: Military psychiatry may give rise to a new type, which one might call "disaster psychiatry"; that may apply to the entire civilian population in the event of atomic warfare. The need for this type of psychiatry is gradually being realized, and I am one of the ardent advocates of a program that will make use of what we have learned in its broader application to civilian groups. Fortunately for that type of planning, we had for study in the Army a rather large cross section of people who had been subjected to considerable stress.

DR. JOSEPH HUGHES: The greatest protection a man has against combat reaction is his capacity for group loyalty.

It is particularly important to establish confidence in leadership, because men cannot develop group loyalties without leadership. The qualities of leadership are present to some degree in 25 of every 100 men; 60 will follow, and 15 will react inappropriately. If the importance of this factor is realized, at least we can have good leadership to offer to the men in the military service and to everyone in universal military training. We can then screen prospective trainees and permit everyone to volunteer for service.

NEW YORK ACADEMY OF MEDICINE, SECTION OF NEUROLOGY AND PSYCHIATRY, AND THE NEW YORK NEUROLOGICAL SOCIETY

Bronson S. Ray, M.D., *Chairman, Section of Neurology and Psychiatry, Presiding*
Joint Meeting, Dec. 12, 1950

Cerebellar Hemangioblastomas. DR. FRITZ CRAMER and (by invitation) DR. WARREN KIMSEY.

The age incidence of cerebellar hemangioblastomas was from 4 to 60 years, with 81.5 per cent occurring between the ages of 20 to 50 years, the average age of incidence being 31. Headache was the first symptom in 91.6 per cent of cases, and its location was equally distributed between the suboccipital and the temporofrontal region.

The postoperative period of freedom from symptoms was longest when all of the tumor or the wall of the cyst was removed. However, when symptoms recurred in cases in which only a cyst had been found and drained at the first operation, there were several instances which proved that the recurrence of symptoms was due to multicentric tumor growths within the cerebellum, and not to a recurrence of the first cyst or tumor alone. In these cases, at subsequent operations the tumors were found to be increasingly angiomatous, whereas the lesion prompting the first operation may have been a simple cyst or one with a nonangiomatous nodule.

This fact seems to be in line with a definite trend toward polycythemia, which was found in 9 per cent of cases before the first operation and in 18 per cent of the entire series of single operations and of multiple operations for recurrences. In the latter group, polycythemia was found in 63.6 per cent. The authors do not subscribe to the theory that the polycythemia is neurogenic in these cases but believe that it arises within the hemangioblastomas themselves in a manner which is as yet undetermined.

In a case of severe polycythemia with necropsy erythropoietic centers were observed in the liver and normal bone marrow. Tissue cultures of cerebellar hemangioblastoma were made from one case; and, although definite evidence of erythropoiesis within the tumor itself could not be substantiated, there was sufficient suggestion of this possibility to warrant further attempts by this method to prove or disprove the possibility of erythropoiesis within the hemangioblastomas of the cerebellum.

DISCUSSION

DR. J. LAWRENCE POOL: First of all, I should like to congratulate Dr. Cramer on his fine presentation. This work has represented a great deal of careful study and observation. Many of these cases are his own, which he has followed and in which he has operated successfully, and a great deal of credit is due him for this careful and stimulating discussion.

There are three or four points I should like to make in regard to this paper. First, there is a greater rarity of multiple lesions associated with hemangioblastoma of the cerebellum than we have been led to suppose. We have all looked for retinal angiomas and seldom seen them; it is justifiable, therefore, to agree with Dr. Cramer that multiple lesions are rare. I question whether the solitary cyst of the cerebellum is always related to this family of hemangioblastomas; I do not feel qualified to pass final judgment on this point, but I tend to question it.

One wonders whether the polycythemia in these cases was a relative, rather than a true, polycythemia, or a reflection of dehydration, which is often intense when such persons are admitted to the hospital because of their nausea and vomiting. The dehydration factor can usually be ruled out, however, because so many patients with tumor of the posterior fossa are brought into the hospital in an acute stage, without polycythemia, but with signs of increased intracranial pressure due to a tumor in the same situation as a hemangioblastoma. That brings up the point whether the polycythemia is neurogenic. I question that because of patients whom we see with tumors of the same magnitude and same location without polycythemia. I believe, rather, that the polycythemia is a reflection of an arteriovenous shunt within these tumors, revealed by histopathologic studies and indicated also by arteriographic studies, as one would expect from the gross and microscopic pathology of these tumors. We know, too, that polycythemia can be associated with arteriovenous anomalies of other types and in other situations in the body. Perhaps further study will indicate that the arteriovenous aspect of these tumors accounts for the polycythemia.

A word of caution as to the diagnosis of polycythemia: One must be careful, by careful blood studies, such as hematocrit readings and plasma volume determinations, to rule out the dehydration factor in reporting this syndrome. Dr. Cramer has been careful to point out that in his cases there is no indication of polycythemia being due to dehydration, for the white cell count, for example, did not indicate dehydration.

Finally, I should like to discuss the belief that angiomas of the spinal canal are another lesion of the same sort, and hence that such patients have multiple hemangioblastomas. I tend to question that belief, for these tumors in the spinal canal seem oftener to be seedings from the original tumor. Patients who complain of pain in the leg of sciatic distribution some time after operation, for example, are found on myelographic study to have multiple tumor nodules of the cauda equina. Dr. Ernest Wood and I are making a study of such patients and find that similar seedings in glioblastomas, as well as in hemangioblastomas, may be demonstrable by myelography. Therefore one must be careful in making the diagnosis of multiple tumor, as the tumors may really be seedings.

X-ray therapy does benefit some patients, but how many it is impossible to say. I base the assertion of the good results of irradiation on the fact that a patient with spinal metastases by seeding from a cerebellar hemangioblastoma had acute sciatic pain and loss of motor power, so that he could not walk, about two years after his operation. A myelogram showed multiple lesions in the spinal canal, for which he was given x-ray therapy. Thereafter he was able to walk and went home. His sciatic pain had also cleared up. That case is virtually proof of a pure culture of this type of tumor in the spinal canal which was relieved by x-ray therapy and is therefore a good index that x-ray therapy may be of benefit in such tumors (slide).

Dr. Thomas C. Guthrie: Since the relation of hemangioblastoma of the cerebellum to polycythemia was first pointed out by Carpenter and Walker (*Ann. Int. Med.* **19**:470 [Sept.] 1943), it has been regarded chiefly as a curiosity. Dr. Cramer has given it statistical foundation by demonstrating a 9 per cent incidence of polycythemia in preoperative cases of hemangioblastoma of the cerebellum, a 57 per cent incidence in cases with recurrence and an over-all incidence of 18 per cent.

Polycythemia has been described in cases of epidemic encephalitis, brain trauma, paralysis agitans and narcolepsy and in 6 cases of brain tumor, each of a different type. In no instance is there an association comparable to that described by Dr. Cramer.

While polycythemia and hemangioblastoma occur together in the same person, there is at present no valid evidence that the destruction of all or part of the tumor reduces the polycythemia. Walker's results are inconclusive concerning this, and operative bleeding and irradiation may lessen an unrelated polycythemia. I should like to ask Dr. Cramer if the polycythemia

seemed to be benefited by surgical measures for an appreciable time. As the tumor progresses and recurs, the hemoglobin concentration and the red cell count increase. The 57 per cent incidence of increase in the erythrocyte count in cases of recurrent tumors supports this observation. In a recent case at Bellevue Hospital, a mild polycythemia developed after the passage of 13 years, finally becoming severe in 17 years, when blood studies revealed 23 Gm. of hemoglobin per 100 cc. and a hematocrit reading of 70 per cent.

The relation of a hemangioblastoma of the cerebellum to polycythemia brings many theoretical considerations to mind, but there is as yet little evidence to support any of them. The disturbance does not seem to resemble polycythemia vera, as the characteristic leukocytosis, thrombocyte increase and hepatosplenomegaly have not been described in any of the cited cases. Whether these cases have the family and racial incidence of polycythemia vera is unknown. Dr. Cramer's autopsy reports illustrate the difficult clinical problem presented by the polycythemic patient who also has many dysfunctions of the central nervous system and papilledema. In cases of this type subdural hematoma and hemangioblastoma are to be strongly suspected. The total number of cases of polycythemia vera with papilledema reported prior to 1949 was only 19, a surprisingly low figure.

Another explanation of the association of hemangioblastoma and polycythemia has been that the polycythemia is due to a cerebral lesion. As Dr. Cramer has pointed out, with other, similarly placed lesions the red blood cell count is not increased. The experimental evidence for this view is not conclusive. It has also been suggested that these cases might indicate the association of a secondary polycythemia with a right to left venous-arterial shunt in the posterior fossa. That many of the neoplastic channels contain blood is entirely in keeping with this view. The secondary polycythemia, which is seen at high altitudes and in association with congenital heart disease, chronic pulmonary disease and hemangiomas of the lung, is found to be associated with a low oxygen saturation of arterial blood. In a case at Bellevue Hospital in the recurrent, histologically verified hemangioblastoma of the cerebellum and severe polycythemia, brachial artery blood was well saturated, with an oxygen content of 26.6 volumes per cent, an oxygen capacity of 27.4 volumes per cent and an oxygen saturation of 97 per cent. In an additional study, preoperative samples of blood were taken simultaneously from the internal jugular vein and the brachial artery. The blood from the internal jugular vein had an oxygen content of 20.1 volumes per cent, and that from the brachial artery, an oxygen content of 22.2 volumes per cent, with an oxygen saturation of 98 per cent. It follows that between the brachial artery and the internal jugular vein there was an arteriovenous oxygen difference of 2.1 volumes per cent, instead of the usual 6 volumes per cent. These determinations seemed to indicate that arterial blood was entering the internal jugular vein, that there was an arteriovenous shunt but that the shunting was not of the type to lower the arterial oxygen saturation and not sufficient to cause a secondary polycythemia.

Dr. Cramer has suggested an original explanation, that the occurrence of hemangioblastomas and polycythemia is part of a widespread vascular change, possibly congenital, in which excessive red blood cell production takes place either in the tumor or in certain organs. Tissue cultures being grown by Dr. Murray suggest that this tumor may have an erythropoietic influence. The case with extramedullary hematopoiesis in the liver, as seen at autopsy, gives the first histologic evidence of the source of the excessive erythrocytosis. This pathologic change, so far as is known, is not observed in polycythemia vera. A study of this familial syndrome, in which cerebellar tumors, retinal tumors, pancreatic cysts, renal cysts, syringomyelia, hemangioblastomas of the spinal cord and polycythemia occur together, may reveal abnormal vascular development. It is interesting that polycythemia has been reported only with the cerebellar tumors.

DR. DANIEL SCIARRA: Has there been a difference in the age of incidence in cases of multiple tumors and that in cases of single tumors?

DR. FRITZ CRAMER: Dr. Pool, I believe, will be interested to know that in the case he discussed there was enough ethyl iodophenylundecylate for a second fluoroscopic examination, and Dr. Wood found that the block had in great part vanished.

We do not have the information respecting the response of the polycythemia to operation which Dr. Guthrie requested. This was an observation which we first made only a few years

ago, many years after we had begun the care of this series of patients. The question of the occurrence of polycythemia with hemangioblastomas in general is one which can be determined more fully only when we are aware of it and make adequate preoperative and postoperative studies of the blood cell count.

Dr. Sciarra, these hemangioblastomas occur and recur at most ages. The average age of onset was about 31½ years, but in our series, and in others cited in our bibliography, there were very young children and there were patients over 60. For the most part, the tumors occurred in the middle decades of life, from 20 to 40 years.

Experimentally Produced Abnormal Mental States. Dr. Paul H. Hoch.

Intravenous injection of synthetic mescaline was used in a series of schizophrenic patients and in "normal" voluntary controls. The changes produced by mescaline in "normal" controls were described. Nondeteriorated patients with overt schizophrenia showed pronounced accentuation of the schizophrenic symptomatology in the emotional and intellectual spheres, with much greater disorganization than was seen in "normal" controls. In pseudoneurotic schizophrenic patients, typical overt schizophrenic pictures were produced, again with more intellectual disorganization and less awareness of reality than in the controls. In chronic deteriorated schizophrenic patients, underscoring was seen in some subjects, but many remained bland, unproductive and burned out after the injection, just as they had been before. Various "therapeutic" measures as applied to the mescaline-produced abnormal mental states were discussed.

Mescaline was also given to a number of patients before and after psychosurgery. It was found that with mescaline it was possible to reactivate the psychosis in patients who improved after psychosurgery but that their response to the drug was quantitatively less conspicuous than before.

DISCUSSION

Dr. Joseph Wortis: Since we are making rather slow progress in determining the etiology of schizophrenia or in developing a truly rational therapy, it may prove easier to study the action of drugs or pharmacological agents which induce schizophrenic-like pictures. There are a considerable number of these "schizophrenogenic" drugs, and one of the most interesting is mescaline. Mescaline, as Dr. Hoch has reminded us, is the active ingredient of a cactus used by the Southwest Indians in ceremonial rituals, and it also seems to be the main ingredient of a Mexican national drink, a distilled beverage called mescal. If we examine the action of this agent clinically and pharmacologically and endeavor to discover its precise action on cerebral function, we can no doubt learn a great deal about the etiology of schizophrenic-like symptoms. Dr. Hoch has made a useful contribution in analyzing and describing these phenomena and in making it clear that mescaline can induce a schizophrenic-like psychosis, especially in predisposed persons, can produce exacerbation of the symptoms of overt schizophrenia and can reactivate a latent schizophrenia in certain cases in which the condition has improved. But, having described that, what can one say further that will be helpful in understanding of the phenomena? It seems to me that the most helpful tack would be to investigate more closely the composition and pharmacological action of the drug. Now mescaline, which is β-3,4,5-trimethoxyphenylethylamine, belongs to a large and varied group of pharmacological agents, the aromatic amines, which include tyramine and several other ethyl amines, and which, according to Quastel and Wheatley (*Biochem. J.* **27**:1609, 1933), have a striking depressant effect on cerebral metabolism. This is nothing new; many years ago studies on brain slices using a dilute mescaline solution showed that the brain metabolism was depressed as much as 30 per cent; with other drugs of the series an even greater depression was noted. A number of other, less intimately related amines have a similarly depressing effect on brain metabolism: indole, skatole and isoamylamine. In this respect, it is interesting to note that mescaline resembles a number of narcotic drugs, the barbiturates, alcohol and probably bulbocapnine, which is an alkaloid and can induce an experimental catatonia, and which is also a cerebral depressant. Could it be that mescaline represents additional confirmation of the Pavlovian hypothesis that schizophrenia can best be regarded as essentially a state of partial cortical inhibition, so that the resemblance that we see so often

between schizophrenic thinking and the thinking that occurs in sleep and dreams may have its common basis in an inhibition of cortical activity? I am sure it is not quite so simple, but it seems to me that emphasis on this factor of cortical inhibition is appropriate.

One might ask: Why is it that mescaline is less effective in cases of lobotomy? Why does it induce a less severe degree of symptoms in these cases? I do not know, and in the presence of Dr. Pool, I can make only the timid suggestion that perhaps it diminishes the total flow of neural stimuli to the cortex—the cortex, so to speak, may live within its means and hence suffer less disorganization in its patterning.

While we ponder these provocative questions, it is good, at any rate, that Dr. Hoch and his associates are proceeding with further experimentation.

I cannot resist the temptation of adding to Dr. Hoch's account some reference to a fascinating subjective account of mescaline written a number of years ago by none other than Havelock Ellis, who took a substantial dose of the drug while alone in his London apartment on Good Friday, 1897 and had a rapturous, but somewhat strange, experience which lasted throughout the night, the details of which are recorded in an essay he published in the *Contemporary Review*, in January 1898. He also gave some mescaline to his friend the poet Yeats, who also recorded his sensations. Interest in mescaline goes back to our American Weir Mitchell, who made some observations on the drug; and, as Dr. Hoch no doubt knows, its effect on psychotic patients was studied as long ago as 1905 by Bresler (*Merck's Rep.* **26**:279, 1905). It is a fascinating drug and may prove to be a very valuable research instrument; I, for one, am grateful to Dr. Hoch for his renewed interest in it.

Dr. Gustav Bychowski: Dr. Hoch is to be congratulated on pursuing experiments which, for unknown reasons, have been abandoned for many years. I wish to add my personal observation with mescaline. Many years ago I had the occasion to observe a very prominent artist, who was both a painter and a writer. He started to take mescaline for his own interest, and he used to paint a great deal under the impact of the drug. Two points are of interest: First, there could be seen in those pictures colors which I never saw again in paintings until this summer, when, while witnessing an Indian ceremonial ritual, I saw the same extremely vivid coloring. There was another phenomenon which the artist had the common sense not to show in his paintings, but which he described, and I should like to know whether Dr. Hoch found it, namely, a good deal of change in body image, mainly in the genitals. He said his genitals appeared to him enormous. I never saw this observation confirmed or described; so I was at a loss to know whether this was due to his peculiarities; perhaps he was sexually preoccupied.

Another phenomenon is the changes in time and space perception which Dr. Hoch observed, but, unfortunately, had no time to discuss. The person I have mentioned experienced these changes both as increase and as decrease. Both time and space seemed able to shrink or to expand tremendously, so that he had images of almost cosmic character, from the beginning of the world, and of tremendous size. This reminds me of observations made in insulin shock, that is, the periodic increase in the internal perception of time. I should like to know whether Dr. Hoch has seen anything similar.

Dr. Leon Rackow: Did the type or severity of the reaction produced by this drug have any prognostic significance in those patients who were later treated with prefrontal lobotomy?

Dr. Paul H. Hoch: Dr. Wortis mentioned many pertinent points. Mescaline definitely depresses the cerebral metabolism, but there are indications that it does not act uniformly in the central nervous system, but that certain parts respond to it as though it is a stimulant and other parts respond as though it is a depressant. Other drugs, such as scopolamine, can produce effects similar to those of mescaline. The difficulty is that other drugs, if given in a higher dose, produce alterations in consciousness, whereas with mescaline all these experiences can be studied in a clear setting, and it is rare that the patient becomes actually confused or shows the organic pictures one sees under the influence of atropine, scopolamine or cannalis (hashish). Mescaline was used by many people in self experimentation, and the literature is full of reports of their experiences. Beringer gave mescaline to practically all assistants in the clinic where he worked, and we have a beautiful description of what they experienced. Their experiences differ somewhat from ours, owing to the fact that they all used mescaline orally and we

administered it as an intravenous injection, which is a much more rapid way of intoxicating the subject. Further, if one takes it orally, it is difficult to know how the person responds to a certain dose.

Dr. Bychowski mentioned the distortion of the body image. That occurs in normal and in psychotic subjects. In the psychotic subject the interesting thing is the bizarre and peculiar interpretations of the body. Such a subject becomes more schizophrenic in his interpretation of the distortions of the body image than the normal person does. We observed several times the presence of alteration of the body image as concerned the genitals, but always in schizophrenic persons. It is interesting that the drug itself has little sexualizing effect, contrary to the action of other drugs.

As to whether the drug has any prognostic significance, the material is not yet assembled, and I do not believe we can answer this question conscientiously. There are two approaches we tried to follow in producing the schizophrenic picture: the physiologic and the psychologic. If one knocks out the ego props of the schizophrenic patient, his time and space perceptions and related functions are much more disorganized than are those of the normal person, indicating that the schizophrenic patient is unable to cope with stimuli in the same way that a normal person does.

Mephenesin (Myanesin®) as a Diagnostic and Therapeutic Aid in Neurology. Dr. Edward B. Schlesinger.

A motion picture was utilized to delineate the following points:

1. The use of mephenesin as a test of prognostic value in the management of the "disk syndrome." The varying responses of significance in evaluating the test were depicted.

2. The use of mephenesin as an aid in determining the static or reversible nature of contractures. Such a technique is useful in planning the nature of therapeutic approach—physical, orthopedic or neurosurgical therapy.

3. An evaluation of the part played by "muscle spasm" in acute anterior poliomyelitis. Evidence is adduced which would tend to minimize the attention focused on this entity.

DISCUSSION

Dr. Byron Stookey: Dr. Schlesinger has demolished the opinion held by many people, those who have contributed to the $13,000,000 fund to which he has referred. I am very glad to see put on a scientific basis the fallacy of the so-called Sister Kenny treatment. It seems to me his film has demonstrated that what is at fault is not muscle spasm, but the pain within the neural arc and that the treatment should be designed to take care of the pain, and not to treat the muscle spasm per se, which is what the Sister Kenny treatment essentially does. Dr. Schlesinger has shown that with complete relaxation the range of motion is not materially increased and that the limitation in movement of the reflex arc is due to pain within the neural arc, and not within the muscle itself. Dr. Schlesinger's demonstration will show that the use of the hot pack, so much emphasized in the treatment of poliomyelitis, is certainly a side issue. Undoubtedly, the hot pack does relieve pain to some extent; certainly it does not affect the basic lesion, and therefore it has definite limitations, which it has been difficult for physicians to put on a scientific basis. Dr. Schlesinger's work, so far as it applies to poliomyelitis, has put it on a scientific basis. As to the effect, I have to revise certain fundamental ideas I have held for years. I should have said that the severe spasticity in the other patients he has shown, such as those with dystonia (I believe one is a patient I sent into the hospital a year or so ago with severe spasticity in both legs, a creature who was operated on without any improvement and with persisting spasticity), was due to the adaptive shrunken periarticular structure and that the lesion was not reversible. Dr. Schlesinger has shown, however, that under the influence of the drug he is able to obtain a range of motion which I would have thought was anatomically impossible. Dr. Schlesinger has shown that the lesion is reversible, and this has tremendous clinical application, in that operations which are designed by the orthopedic surgeons to lengthen tendons and to stretch periarticular structures (a procedure which Dr. Schlesinger says is actually tearing them) should not be done until it has been determined what range of motion

can be obtained under the influence of the drug which Dr. Schlesinger is using. Certainly, it will put the treatment of such contractures on a scientific basis, so that in these two respects I I think Dr. Schlesinger's studies are of tremendous value. Certainly, where so much effort is being made to treat patients with physical therapy, when physical therapy has actually only about the same effect as putting a patient with a high temperature in a cold pack and taking him out of it, and having the temperature go back to where it was before the pack was given, Dr. Schlesinger has laid a scientific foundation for the interpretation of muscle spasm and of the effect of the neural arc in the maintenance of motion.

Dr. Rollo J. Masselink: I should like to cite another use for this drug, and this as of Thursday of last week. I saw a patient who was called hysterical and who had paraplegia in flexion. I made a tentative diagnosis of meningioma or neurofibroma at the fourth thoracic segment. I wanted to make a myelogram, but the patient's legs were pulled up on her abdomen and could not be moved. Dr. Schlesinger gave her 100 cc. of mephenesin. We were able to straighten her legs out on the x-ray table and take a myelogram. This would have been impossible without the drug. We found the lesion at the third thoracic level, operated on her today and removed a meningioma. This demonstrates the practical application of a drug of this sort.

Dr. Daniel Sciarra: Did the woman have pain during the taking of the myelogram?

Dr. Rollo J. Masselink: No.

Dr. Daniel Sciarra: Dr. Schlesinger's film speaks for itself as regards the beneficial effects of this drug. I would like to make a few general comments, since Dr. Schlesinger points out that this film covers a small ramification of muscle relaxants. I suppose the aim of muscle relaxants is to relax the muscle without undesirable side effects. I am not sure that by this definition mephenesin would be called a good muscle relaxant. It would be more correctly designated a muscle paralysant or a drug that causes paralysis of muscle. In the course of its paralyzing action, Dr. Schlesinger has been keen enough to pick up some uses for this potentially toxic drug. He has indicated that we do not have good muscle relaxants for oral use, and I am not sure that we have a good muscle relaxant to give by vein. Mephenesin has effects similar to those of a mild anesthetic which will cause paralysis of muscles, and in the course of that action he could carry out maneuvers which are of valuable prognostic significance.

I should like to make some points in relation to the disk syndrome. Dr. Schlesinger has indicated that it is valuable to use mephenesin in cases of the syndrome as a prognostic test. I think he will agree that this does not indicate the eventual prognosis for the patient. It merely indicates the prognosis for that particular attack, whether it will respond to conservative therapy or whether the patient will have to be operated on for relief of the pain. However, the same patient may eventually have another attack of pain which will necessitate operation. I do not know whether Dr. Schlesinger has cases which have belonged to group 1 and have shifted to group 3. If he has, they would illustrate this point.

In connection with Dr. Masselink's point about hysteria: There have been attempts to show the use of mephenesin in differentiating neurologic and psychiatric disease. I think that differentiation is not helped by mephenesin. The drug will act in a nonspecific fashion; the patient's muscles will become relaxed or paralyzed, and the differentiation of psychogenic and neurologic disease is not possible.

The last point is in relation to spasticity. Dr. Schlesinger has amply demonstrated to those of us who are fortunate enough to work with him some of the implications of the use of mephenesin. I should like to make a plea in connection with a very common syndrome, that of spastic hemiplegia as a result of cerebral thrombosis. Often one is told that the patient is not going to respond to physical therapy. However, after months, and even years, of the presence of a hemiplegia, the mephenesin test has demonstrated certain reversible phenomena that make additional physical therapy feasible and practical.

Dr. Edwin A. Weinstein: I want to call attention to one other aspect. Most of the discussion so far has concerned the action of mephenesin on the muscle, on spasm and on the motor pathway. It seems to me that Dr. Schlesinger, like Dr. Hoch, has described an experimentally produced abnormal mental state. He said the drug took effect when nystagmus appeared, indicating that it was acting on the brain. I think he produces a state of euphoria,

an alteration of consciousness in which the perception of pain is altered, and that is an extremely important factor in diminishing the pain and spasm. Frequently patients with subarachnoid hemorrhage who are paranoid or euphoric say they have no headache, and no nuchal rigidity is demonstrable. To stress the point again, the action is on the entire nervous system, and not on the muscles alone.

DR. PAUL H. HOCH: There is no doubt that mephenesin has a strong central action. Nevertheless, I believe that what Dr. Schlesinger demonstrated cannot be explained by the central action of the drug. The muscle relaxation does not take place because the patient becomes euphoric. We see muscle relaxation in patients in whom the central action of the drug produces depression, which usually reenforces the muscular tension. Dr. Schlesinger mentioned that the oral application of the drug is ineffective. Mephenesin is now widely publicized as a drug which relieves anxiety. In our hands, it reduced anxiety rarely, and not effectively. The studies with anxiety-controlling drugs will have to be much more carefully controlled to eliminate suggestive influences.

DR. BYRON STOOKEY: I should like to add one word in objection to what Dr. Masselink suggested, that the drug be used in the differential diagnosis of hysteria. There is a much better drug that that which Dr. Kennedy has used in the past; he may have forgotten it, but he used it: A pint of whisky is much more valuable in differential diagnosis; if that is given, the patient who was previously paralyzed gets up and walks. I should like to put in a plea for the use of alcohol, and not mephenesin, in the differential diagnosis of hysteria.

DR. ROLLO J. MASSELINK: I did not suggest this as a differential means in the diagnosis of hysteria. The woman was called hysterical before we saw her. I was only suggesting another use of mephenesin: We could not do a myelographic study on this patient with her legs up on her abdomen.

DR. EDWARD B. SCHLESINGER: I should like to thank Dr. Hoch for confirming my position in the matter of the oral use of mephenesin. You may realize from his words why I was so repetitious about disavowing any belief in the oral use of the drug. I have always felt quite differently from Berger, who widely disseminated the concept of the usefulness of mephenesin given orally.

Let me reiterate that what I have said about the drug as a prognostic test relates only to the attack of acute low back pain and the intervertebral disk syndrome. It is obvious that one cannot make any prediction as to what further degenerative changes or trauma may occur. We are trying to use the test to find out whether the patient will come to operation during this attack or will recover after conservative measures.

I may add to Dr. Hoch's point, that is, that the effect of the drug is not purely on the psyche, by pointing out that the drug has obvious effects on many different portions of the central nervous system. These include mechanisms in the cerebrum, brain stem and cord segments. In the patients we have discussed, I believe that the primary effect is at the bulboreticular level. I also feel that what we see is not an emotional response at all, but a physiologic change in facilitation—inhibition mechanisms at the level of the bulboreticular substance.

News and Comment

INTERNATIONAL CONGRESS FOR PSYCHOTHERAPEUTICS

Under the auspices of the Dutch Society for Psychotherapeutics, an International Congress for Psychotherapeutics will be held at Leiden Oegstgeest (Netherlands) from Sept. 5 to 8, 1951, inclusive. Sections will include papers on child psychiatry, group psychotherapy and psychosomatic therapy. There will be opportunity for foreign speakers to be placed on the program.

Inquiries should be addressed to A. H. Fortanier, M.D., secretary of the International Congress for Psychotherapeutics, Psychiatrische Cliniek, Der Universiteit Leiden te Oegstgeest.

PSYCHIATRIC TRAINING FACULTY OF MASSACHUSETTS, INC.

The Psychiatric Training Faculty of Massachusetts, Inc., announces that applications are being accepted for residency training in psychiatry and neurology. Residency salaries are available which permit immediate commitment to applicants who have satisfactorily met the requirements of the Faculty. The following affiliated units are utilized in planning the training program for each individual resident: Boston Psychopathic Hospital, Boston State Hospital, the Judge Baker Guidance Center, Massachusetts General Hospital, Massachusetts Memorial Hospitals and Worcester State Hospital. Opportunities are also afforded for psychoanalytic training for a limited number of candidates under the auspices of the Boston Psychoanalytic Institute. Arrangement for such training is dependent on acceptance by the Institute. Applications should be made on a form secured from the Psychiatric Training Faculty, Room 800, 15 Ashburton Place, Boston 8.

CORRECTION

In the article by John R. Blake Jr., M.D., and E. Charles Kunkle, M.D., entitled "The Palmomental Reflex: A Physiological and Clinical Analysis," published in the March issue of the ARCHIVES, page 337, the sentence beginning on line 7 of page 341 should read, "The response in four of the subjects was reduced or abolished coincident with the onset of moderate impairment of 'fast' conduction pain sensibility, despite the accompanying dysalgesia of 'slow' conduction pain induced by stimulus."

Obituaries

OLIVER SMITH STRONG, Ph.D.
1864-1951

When Oliver Strong died on Feb. 22, 1951, there passed one of the last of a remarkable group of American neuroanatomists that did so much to advance the science of neurology in this country. They were primarily comparative anatomists, but what they taught was as much human as it was comparative neurology. All were teachers, and Dr. Strong was one of the most inspiring of the lot.

Oliver Strong was born in Red Bank, N. J., on Dec. 30, 1864. He was educated in private schools before going to Princeton, where he received an A.B. degree in 1886 and an A.M. degree in 1888. For one year he was assistant in biology at Lake Laboratory in Milwaukee before he went to Columbia University, where he taught neuroanatomy and histology continously until his retirement in 1937. At first he taught in the department of zoology, but after 1904 he spent all of his time at the College of Physicians and Surgeons of Columbia. In 1895 he had earned the degree of Doctor of Philosophy.

It was with the College of Physicians and Surgeons that Strong was almost exclusively associated, first as assistant in histology, when he did most of the teaching in neurohistology. In 1917 he was appointed assistant professor of neurology, and in 1927 he was raised to the rank of professor of neurology and neurohistology, which position he held until he retired in 1937.

Dr. Strong contributed the chapter on neurohistology in Bailey's "Text-Book of Histology," which was later published as a separate volume and then elaborated, in collaboration with Adolph Elwyn, as "Human Neuroanatomy" (1943).

Dr. Strong was an honorary member of the American Neurological Association and of the American Association of Anatomists.

Strong's earliest published article was on the cranial nerves of amphibia, but later in life he wrote and published very careful observations in the field of clinical neurology in the Archives of Neurology and Psychiatry, as well as in other medical journals. For years he was associate editor of the *Journal of Comparative Neurology* and later was an honorary member of the editorial board. When the *Bulletin of the Neurological Institute of New York* was established, Strong did most of the editorial work, and practically all of it from 1930 to 1932, when the publication was suspended.

Teaching was ever Oliver Strong's forte. His great knowledge of neuroanatomy and his strict scientific honesty made him the kind of wise mentor younger men relied on. Sternly critical of himself, he was just as critical of the rest of us, and we learned much of value from him besides neuroanatomy. The department of neurology at the College of Physicians and Surgeons, over which Frederick Tilney presided so well, owed just as much to Oliver Strong, who started Tilney on his career as a neuroanatomist. In the old Vanderbilt Clinic, Strong was always with us with a peculiar title of consultant in neuroanatomy. He worked with us over clinical material and with the meticulous care with which he did everything. It was principally the latter characteristic that made him such an inspiring teacher. In the twilight that descended on him in his later years we lost personal contact with him, but his inspiration and spirit still is a living thing for those of us who knew and loved him.

Louis Casamajor, M.D.

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